TRANSACTIONS

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and
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ANNUAL MEETING OCTOBER 12-17, 1952, CHICAGO

WIDE APPLICATION IN OPHTHALMOLOGY

Neo-Synephrine hydrochloride, because of its dependable vasoconstrictive and mydriatic actions, has many indications in ophthalmology

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⅛% solution.
For rapid relief of congestion, itching, smarting and lacrimation.

REFRACTION, FUNDUSCOPIC EXAMINATION:
2.5% solution or 1% emulsion.
For prompt and short acting mydriasis virtually free from cycloplegia.

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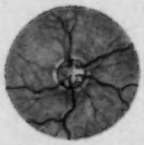
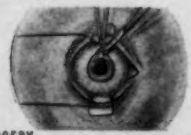
10% solution or 10% emulsion.
For freeing recently formed posterior synechiae as well as for prevention of synechiae formation in uveitis (with atropine).



GLAUCOMA (certain cases and tests):
10% solution, 10% emulsion or 2.5% solution.
For temporary reduction of intra-ocular tension and for the provocative test for angle block.



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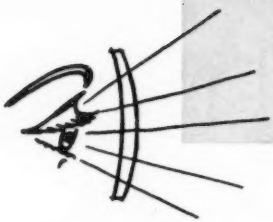

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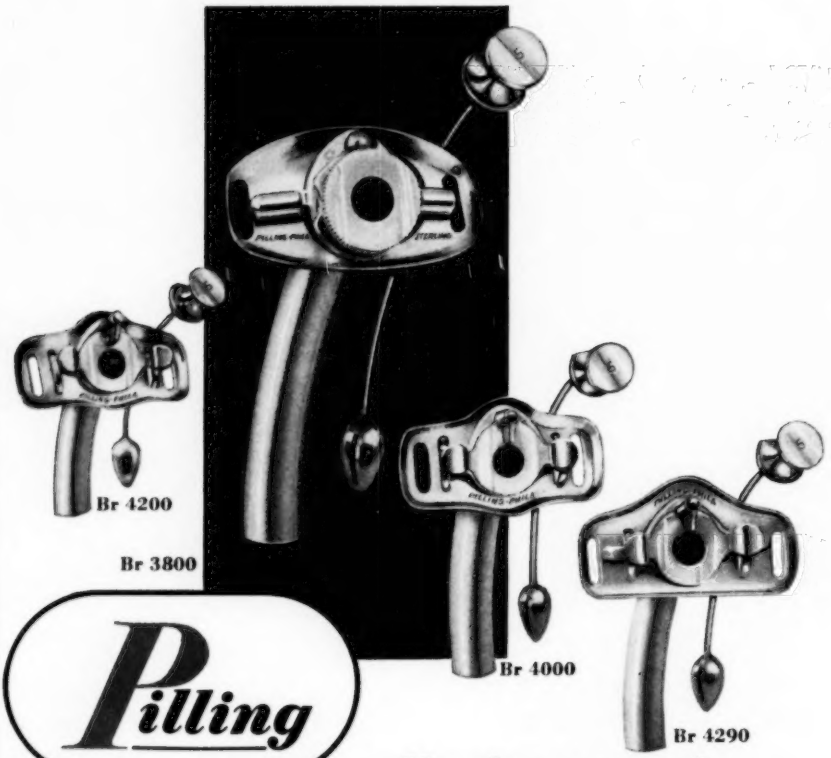

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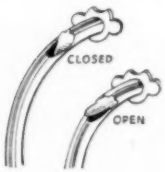

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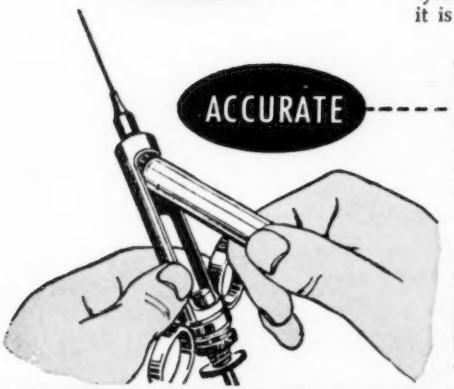

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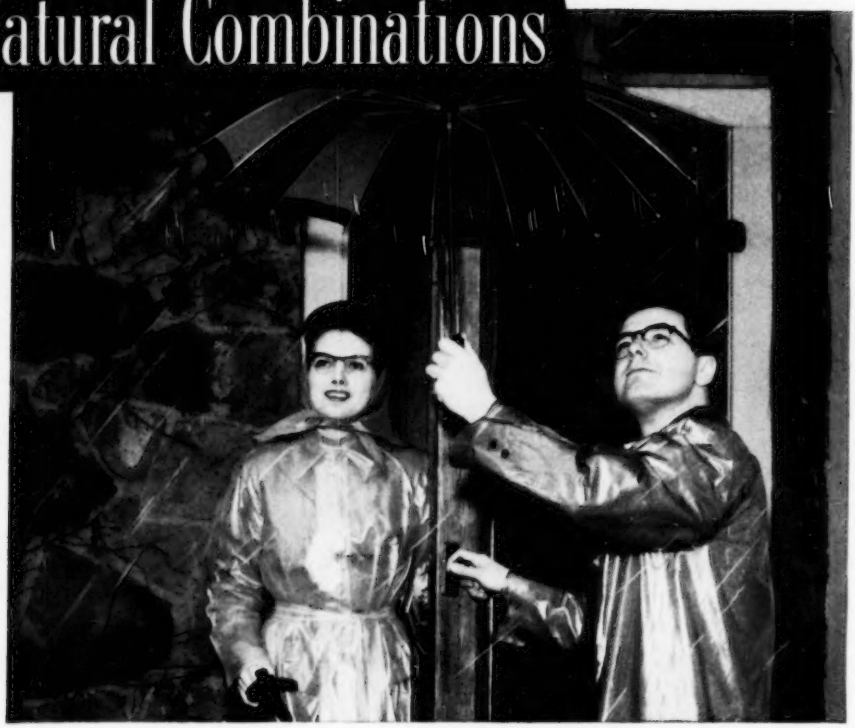

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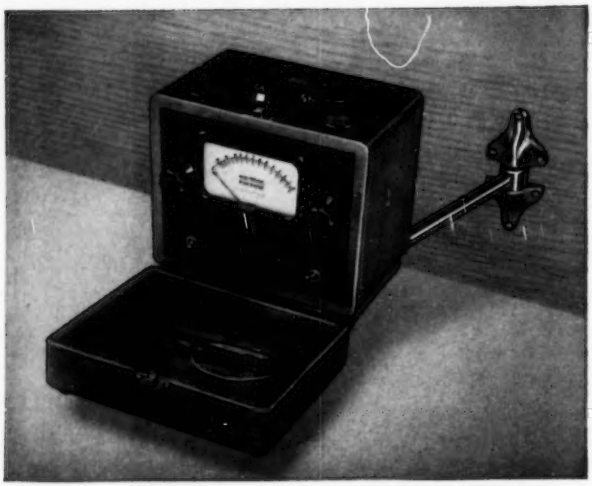

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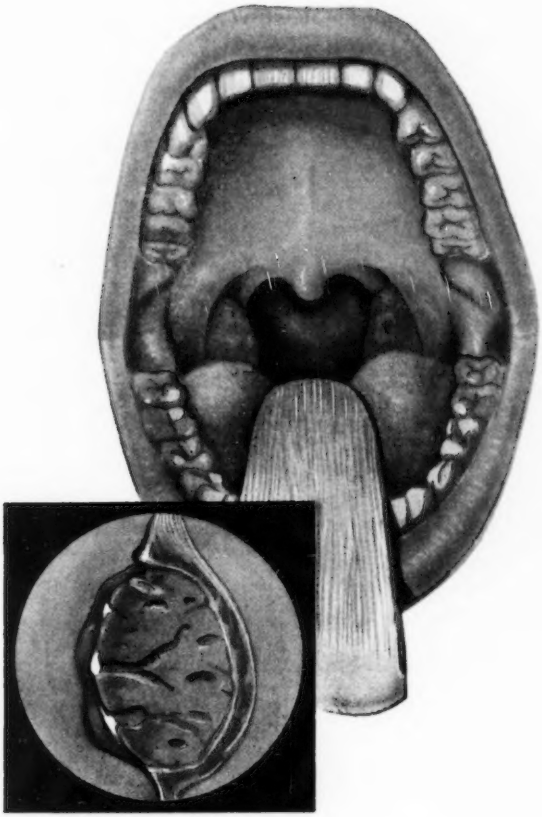

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Sayer, R. J.; Michel, J.; Moll, F. C., and Kirby, W. M. M.: Am. J. M. Sc. 221:256 (March) 1951

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* Reiter, Carl: A New Synergistic Mydriatic Combination, A.J.O. 35:412-13, March 1952.

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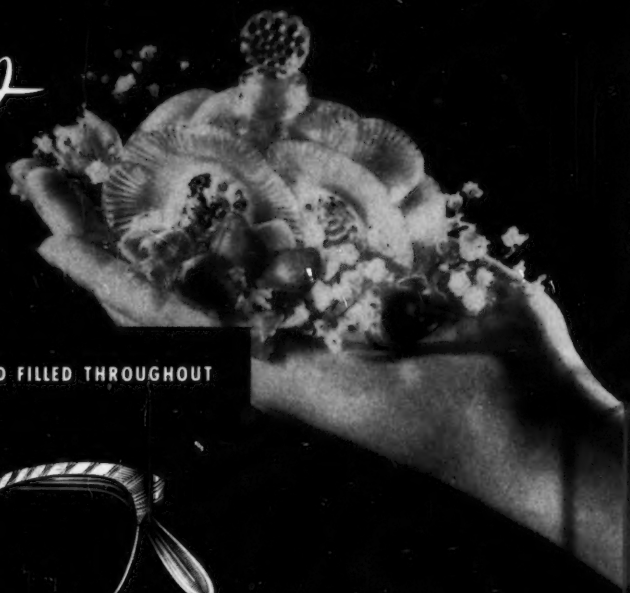

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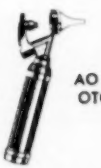

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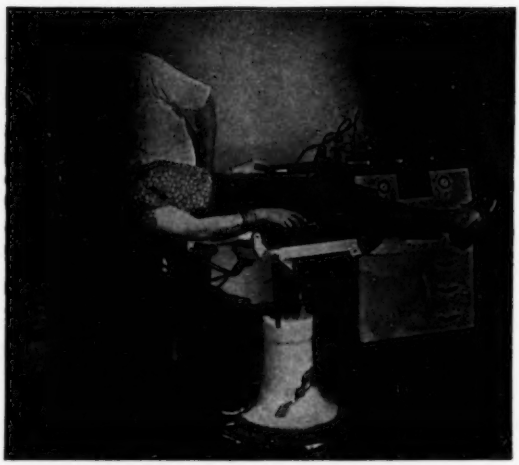

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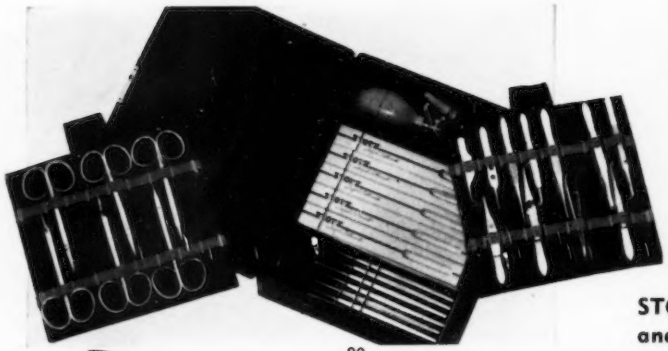

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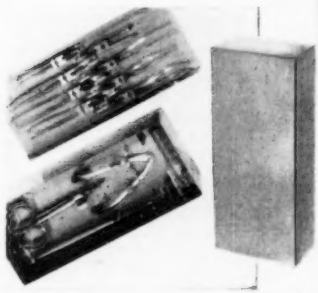

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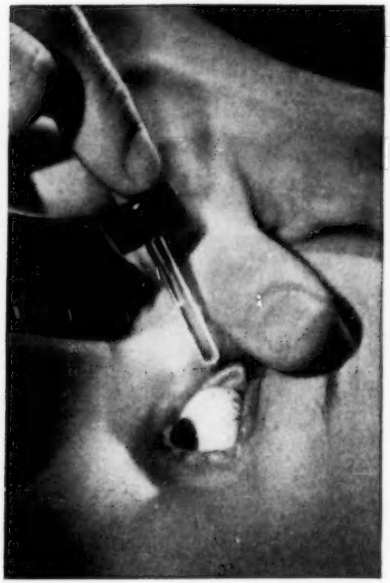

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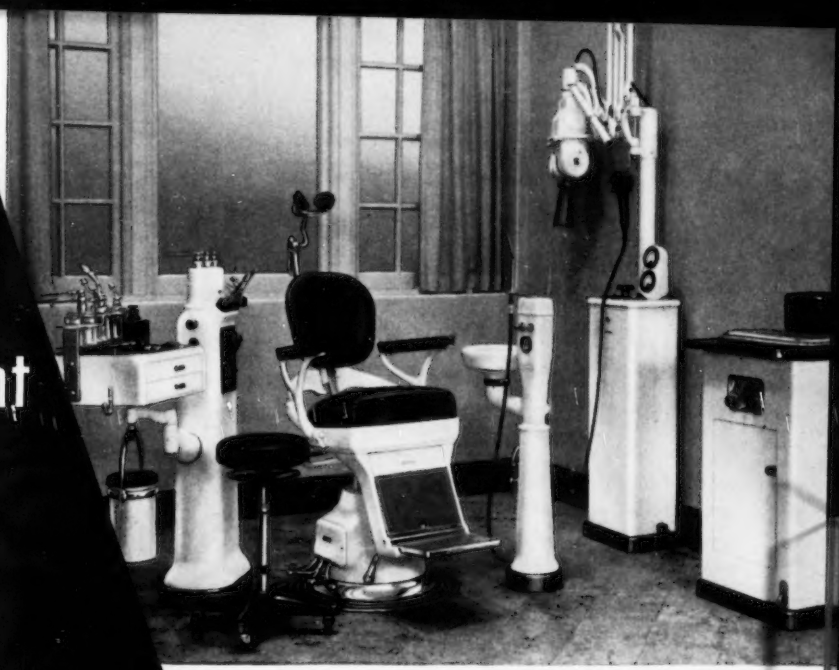

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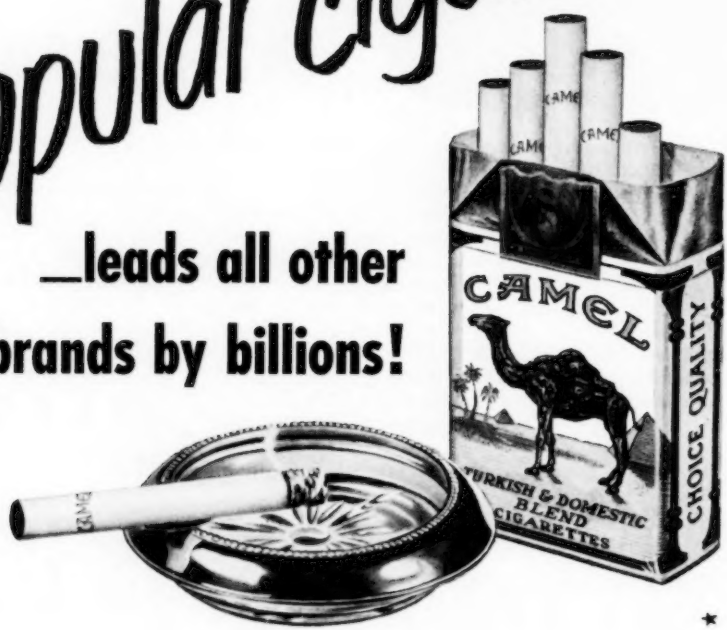

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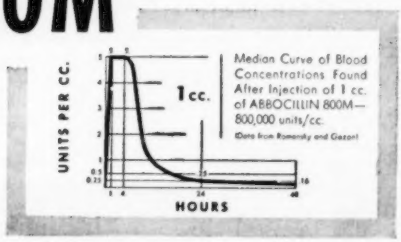
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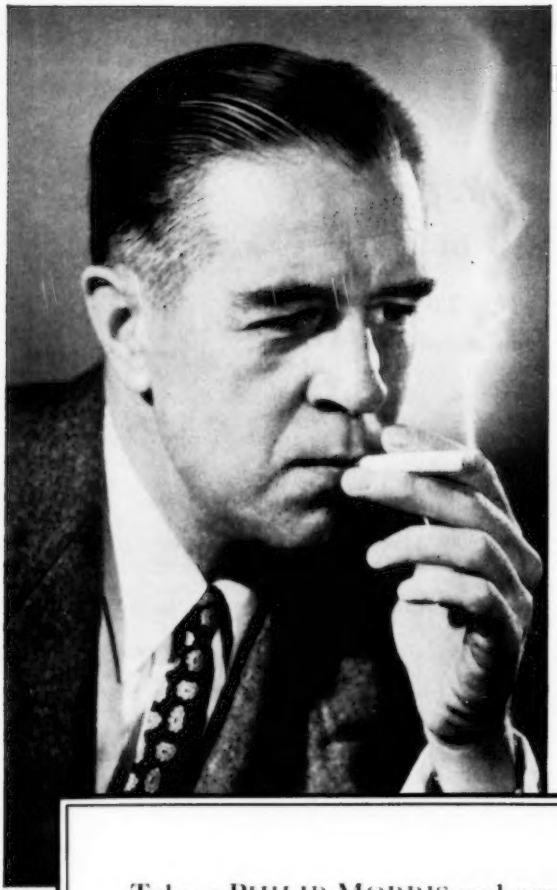

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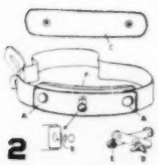

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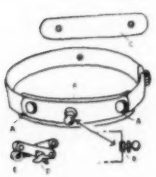

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THE PATHOLOGY OF THE NOSE AND PARANASAL SINUSES IN RELATION TO ALLERGY

With Comments on the Local Injection of Cortisone

HERMAN SEMENOV, M.D.
BEVERLY HILLS, CALIF.

INTRODUCTION

IT was not so long ago, and yet the time seems distant, when all rhinitis and sinusitis was attributed to infection, when words such as histamine and allergy were known only to research workers, and Sir William Osler called syphilis "the great imitator." As matters now stand, "the great imitator" seems to be allergy itself.

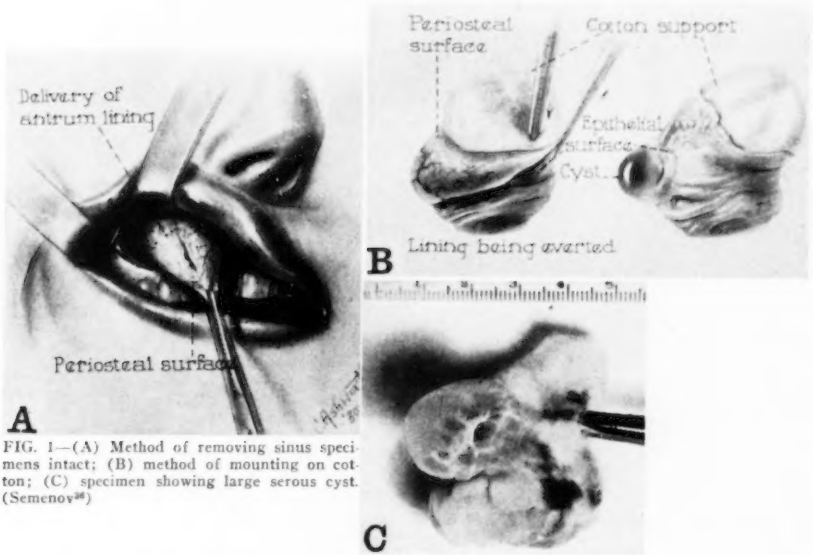

FIG. 1—(A) Method of removing sinus specimens intact; (B) method of mounting on cotton; (C) specimen showing large serous cyst. (Semenov[26])

In any consideration of the pathology of the nose and sinuses, we must reckon with allergy, not only its typical form such as hay fever but also its masked form as in "collagen disease." In some of these obscure conditions, infection is the precipitating factor. The storm of devastation that follows, however, rotates in allergy and often runs a relentless up and down course, which we fought for years under the tattered and torn banner of focal infection.

My interest in allergy goes back to 1928, when the focal infection theory was at its height and Frank B. Kistner reached into the paranasal sinuses and skilfully withdrew diseased membranes in such a manner that they could be studied in one whole piece and in every detail (fig. 1).

Attending Otolaryngologist, Cedars of Lebanon Hospital, Los Angeles, Calif.
Presented in part at the meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, Oct. 19, 1951, Chicago, Ill.

At that time, allergy was in its infancy and only a handful of workers were interested in it, among others, Robert L. Benson,[1,2,3] Professor of Pathology at the University of Oregon, with whom I worked under a research fellowship. When Kistner[15,16,17,18] asked for help, the obvious thing to do was to work out the normal histology; and, in the following pages, I shall go back to the beginning and republish some of our original photographs.

The material, as stated in previous publications,[36] includes more than a thousand specimens. The entire membrane often was removed intact by careful subperiosteal dissection (Caldwell-Luc, and external frontal-ethmosphenoidal surgery) and mounted on supports (cotton) (fig. 1,B). The bacteriology of 400 sinuses was investigated by making swabs and grinding a portion of the tissues in sterile sand. A representative collection of this material (approximately 200 specimens) has been placed in the Registry of Otolaryngic Pathology at the Armed Forces Institute of Pathology (AFIP) along with many of my negatives. Some of this material will appear in the forthcoming Atlas of Otolaryngic Pathology, and also in the Manual of Applied Pathology being prepared for the Home Study Courses. The present manuscript is one chapter of that manual. A picture, it has been said, is worth a thousand words. With this in mind, let us begin with the sinuses because the histology is less complicated in these cavities.

APPLIED HISTOLOGY

In contrast to the nasal mucous membrane, whose familiar color attests to the rich blood supply that it takes to warm and moisten the inspired air, the lining of the paranasal cavities is comparatively thin. On entering the sinuses, it becomes so attenuated that large areas contain very few, if any, mucous glands, and the histology virtually is reduced to a layer of epithelium and a layer of connective tissue. The thickness of the lining varies somewhat and usually is less than half of a millimeter under normal conditions.

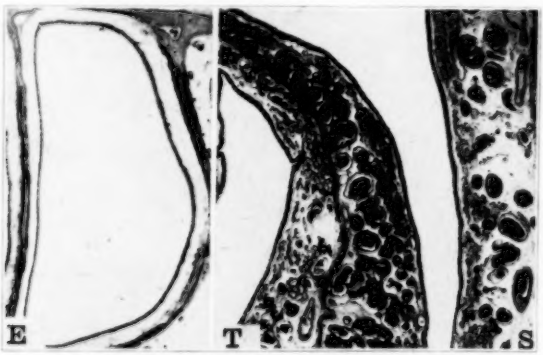

FIG. 2.—Comparison of the thickness of nasal and sinus mucous membranes (magnified 10 X). (E) ethmoidal cell. (T) middle turbinate. (S) nasal septum. Note the epithelial line and compare the tunica propria in each example. "T" and "S" contain many glands and are extremely vascular. "E" looks pale by comparison. (Semenov[36])

The nasal mucosa, on the other hand, is much thicker and has a richer supply of blood vessels and glands, and a greater abundance of stroma (fig. 2), which naturally varies from place to place, depending upon the anatomy and function of the part. It is very thin, for example, in the upper part of the lateral nasal wall (ethmoidal area) (fig. 3), and in this location bears a striking resemblance to the lining of the sinuses. This is the area in which we so often encounter nasal polyps, and there can be little doubt that the histology (delicate tunica propria) favors polypoid degeneration.

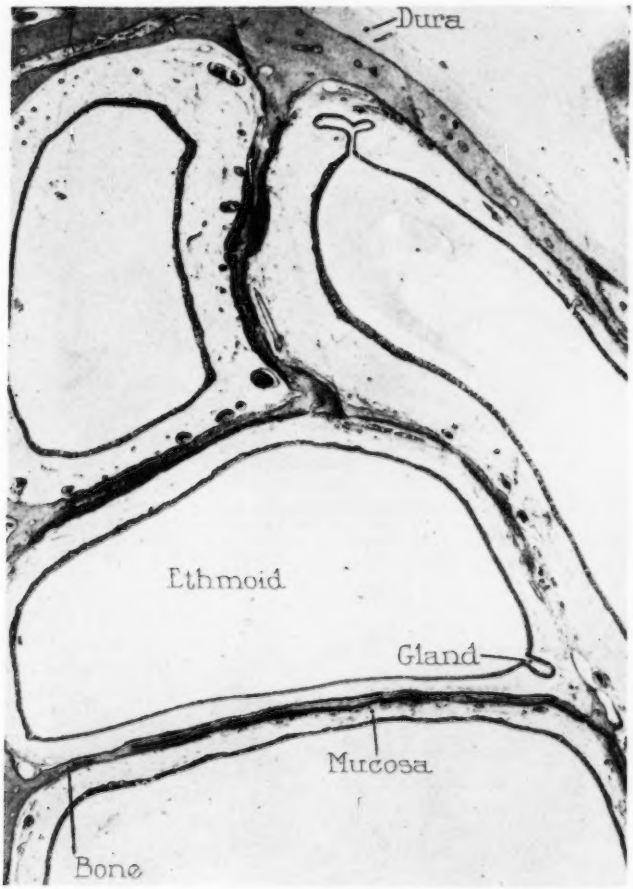

FIG. 3—Cross section of ethmoidal capsule (magnif. 18X). On the left are two ethmoidal cells (posterior ethmoidal sinuses) and below them, half of an anterior cell. On the right are the spheno-ethmoidal recess and roof of the nasal cavity. The character of the mucous membrane in the sinuses and uppermost part of the nasal cavity is similar in this location. Note the smooth clean appearance of a perfectly normal specimen. (Semenov[35])

It is interesting to read in this connection the views of Alexander Monro published in the year 1783, in "A System of Anatomy" by Monro, Winslow, and Innes.

Though the membranes, which line the frontal, sphenoidal, and maxillary sinuses, are continuations of the one which covers the bones within the nose; yet they are much thinner than it is, and have so much smaller vessels, that the injection which makes the membrane of the nose red all over, fills only some few vessels of the maxillary sinuses, and scarce is observed in the frontal and sphenoidal. Are not the larger vessels intended for a more plentiful secretion of a viscid liquor to defend the membrane from the effects of the perflatus, which is constantly through the nose? Are not the membranes which have the smallest vessels, *cae-*

FIG. 4—A normal sinus mucous membrane (A) magnified 5X compared with a hyperplastic one. (B) Sphenoidal sinusitis. (Semenov[36])

FIG. 5—Chronic hyperplastic purulent sinusitis (magnif. 5X), thickened from ten to forty times normal. Compare thickness with figure 4 (A) and note polyps and crypts, as well as the prominent edema (lighter areas). (Semenov[36])

FIG. 6—Chronic allergic sinusitis (magnif. 5X). Compare the thickening with figure 4 (A) and note the polypoid swellings, mass of mucus on the left and dark shadow in the subepithelial region, which marks a dense infiltration of leukocytes and eosinophils. (Seminov[36])

TABLE I

NORMAL AND PATHOLOGICAL THICKNESS OF SINUS MEMBRANES

	NORMAL VARIATION IN MILLIMETERS	PATHOLOGIC THICKENING IN MILLIMETERS
Maxillary sinus		
Medial wall	0.3 —1.0	1.0—20.0
Lateral wall	0.1 —0.5	1.0—20.0
Frontal sinus	0.07—0.3	1.0— 4.0
Ethmoidal sinus	0.08—0.4	1.0— 5.0
Sphenoidal sinus	0.07—0.5	1.0— 5.0

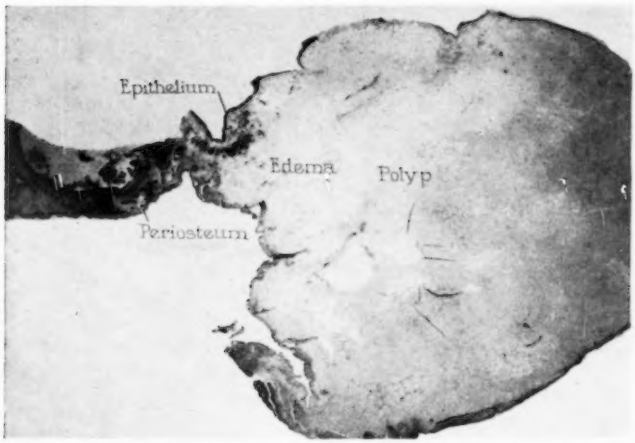

FIG. 7—Polyp in the maxillary antrum. The great increase in thickness is largely due to pathologic changes in the connective tissue of the tunica propria. (Semenov[25])

teris paribus, the most sensible? Are not many phenomena of smelling, inflammations of these parts, megrim [migraine], polypi, etc., depending on this structure of these membranes?

CONNECTIVE TISSUE

The great anatomists knew much more than we sometimes realize, and in an age of electron microscopy and "collagen diseases" we are just beginning to appreciate the tunica propria (L., *proper coat)* of a mucous membrane and the connective tissue substances that it contains. When we measure and compare the pathology in different parts of the nose and sinuses, we cannot escape the conclusion that the character of a mucous membrane depends, in no small way, upon the development of its tunica propria.

The tunica propria is the layer of stroma that tears so easily during a submucous resection of the septum, and the perichondrium or periosteum is the tough one, even though it is much thinner. In chronic sinusitis the mucous membrane tends to become hyperplastic and sometimes reaches a thickness that may be ten, twenty, thirty and even fifty times greater than normal (figs. 4, 5, 6, 7). In almost every case, the bulk of the pathological thickening centers in the mesenchymal substance of the tunica propria.

Histological sections often show a sharp line of demarcation between the tunica propria and periosteum, in fact, we may identify three distinct layers of stroma: (1) basement membrane, (2) tunica propria, (3) periosteum. Each of these layers has certain peculiarities of its own, and during inflammation, particularly allergic inflammation, the differences begin to stand out (fig. 8).

Basement Membrane

In perfectly normal specimens, the basement membrane is so thin that it is practically invisible. With thickening of the basement membrane, an easily recognized pink-staining hyalin layer appears directly under the epithelium. It is composed of delicate collagen fibrils (reticulum) and, in chronic allergic rhinitis and sinusitis, the collagen undergoes marked hyalinization and swelling which may reach a thickness of 20 micra or more. A very thin layer of

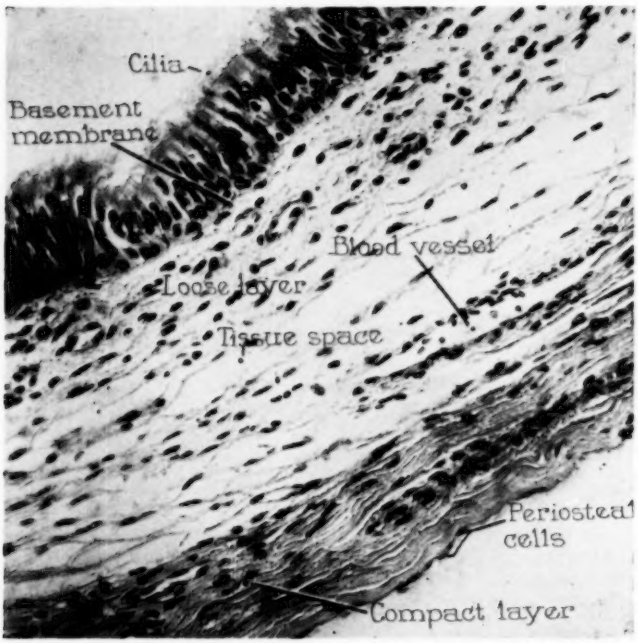

FIG. 8—Histology of the connective tissue layers in a sinus mucous membrane. The basement membrane is almost invisible. The loose layer of connective tissue is the tunica propria, and the compact layer is the periosteum. A slight edema is present which brings out the difference between these two layers. The epithelium is nearly normal. Mild inflammation is present as indicated by the edema and increased cellularity of the tunica propria. The total thickness of this membrane is 0.363 mm. (Semenov[25])

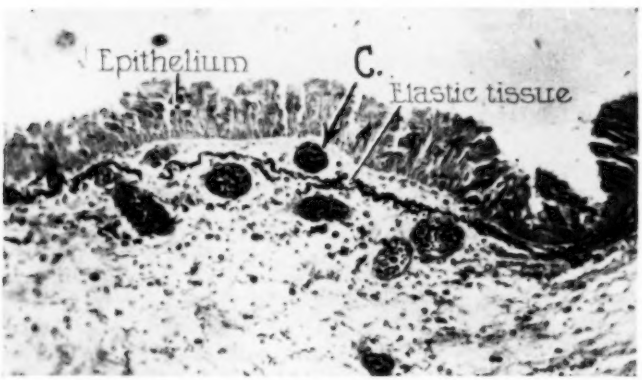

FIG. 9—Subepithelial region of a sinus mucous membrane showing elastic tissue fibers lying just below the basement membrane. The basement membrane itself is composed of reticular fibrils which blend into the tunica propria. (C) is a capillary lying between the basement membrane and the layer of elastic tissue fibers. (Semenov[25])

elastic fibers may occur just under the basement membrane (fig. 9), but it is not constant.

Tunica Propria

The tunica propria of the sinuses is composed of delicate lamina of loose connective tissue. It contains a great many interstices and areolar tissue spaces which, like a sponge, fill with great quantities of fluid and blow up like a balloon on the slightest provocation. The thin collagen fibers and fibrils that make up this delicate layer of stroma rupture easily (fig. 10) and permit a peculiar cystic condition (serous cyst) to form in the antrum. Half an ounce of clear amber-colored fluid (plasma) may be recovered from a single cyst. No other mucous membrane can boast of such vast accumulations of fluid, and no other membrane can become more edematous.

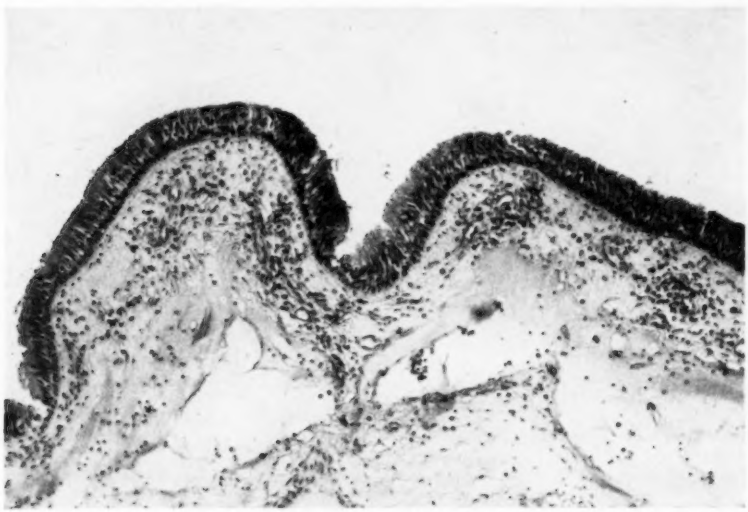

FIG. 10—Chronic allergic sinusitis showing the intense edema and its effect on the delicate fibrils of the tunica propria. Large pockets of fluid often occur and form the polypoid swellings that characterize this condition. Note the rupture of the tissue spaces, broken fibers and edema fluid. (Van Alyea and Semenov[9])

Fibroblasts (fibrocytes) are the main cells of connective tissue. They are long, flat or spindle-shaped. The nucleus is large and oval and has delicate chromatin particles (fig. 11).

Many histologists believe that *undifferentiated mesenchymal cells* persist even in adult life. These cells resemble fibroblasts but are smaller and are found along blood vessels or capillaries. They are supposed to furnish new cell types.

The *histiocytes* are almost as numerous as the fibroblasts, and are scattered among them. In inflammation they become ameboid and are called macrophages. They may be rounded or spindle-shaped, and often look like fibroblasts. According to Fenton and Larsell[8] "these cells are the first line of defense and repair, appearing ahead of the polymorphonuclears and the lymphocytes." They have been called pericytes, clasmatocytes, adventitial cells, resting wandering cells, and many other names, and appear in almost all connective tissue.

Lymphoid tissue begins to appear with inflammation and exhibits all variations from small macrophages (polyblasts) to typical lymphocytes, as seen in the blood stream. These cells can form in the loose connective tissue itself, although many come in from the blood

(hematogenous), and one cannot be separated from the other because of the constant exchange of cells.

As stated previously, *the tuncia propria of the nasal mucous membrane* varies from place to place; in certain areas it is thick and highly developed and in others it is thin and delicate.

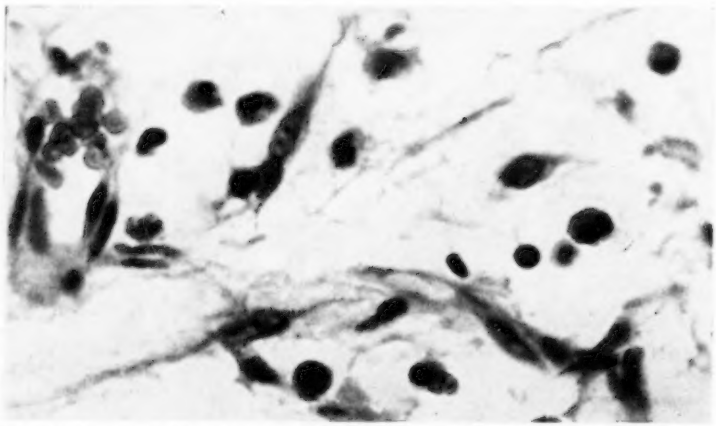

FIG. 11—Young fibroblasts in an allergic polyp. Note the capillary and red blood cells on the left, infiltration of eosinophils (bilobed cells), lymphocytes and plasma cells. The connective tissue is edematous and has a "myxoid" appearance. (810 X).

TABLE II

THICKNESS OF THE TUNICA PROPRIA OF THE NOSE

	MILLIMETERS
Ethmoidal Capsule	0.2
Middle meatus	0.3
Nasal septum	0.8
Turbinates	
Superior	0.3
Middle	1.0
Inferior	2.0

The turbinal tissues are very thick and complicated, owing to the abundance of glands and cavernous blood vessels. These organs naturally require a well designed supporting stroma owing to the constant strain that is put upon them. Without going into details, it is only necessary to point out the important differences: (1) the superficial (subepithelial) stratum has a reticular layer which, on the whole, is less areolar than that of the sinuses, (2) the glands and erectile tissue lie below this layer and are embedded in a tougher stroma, (3) a deep stratum of compact collagen fibers is present, and this layer is fused to the underlying periosteum. The subepithelial layer is the one that suffers the most inflammation, and considering the terrific beating that it takes almost every day of our life, it holds up very well.

The loose connective tissue of the tunica propria serves several functions:

1. It serves as a packing material and supporting stroma.
2. It plays an important part in the nutrition of the overlying epithelium.
3. It may have an endocrine function, but the evidence supporting this view is not complete.
4. Of great importance is its role during inflammation, and a conspicuous part is played by the macrophages.
5. The qualities of the loose connective tissues make up the "constitution" of an individual, and the allergic or hypersensitive state is manifested by alterations of the loose connective tissue.[23]

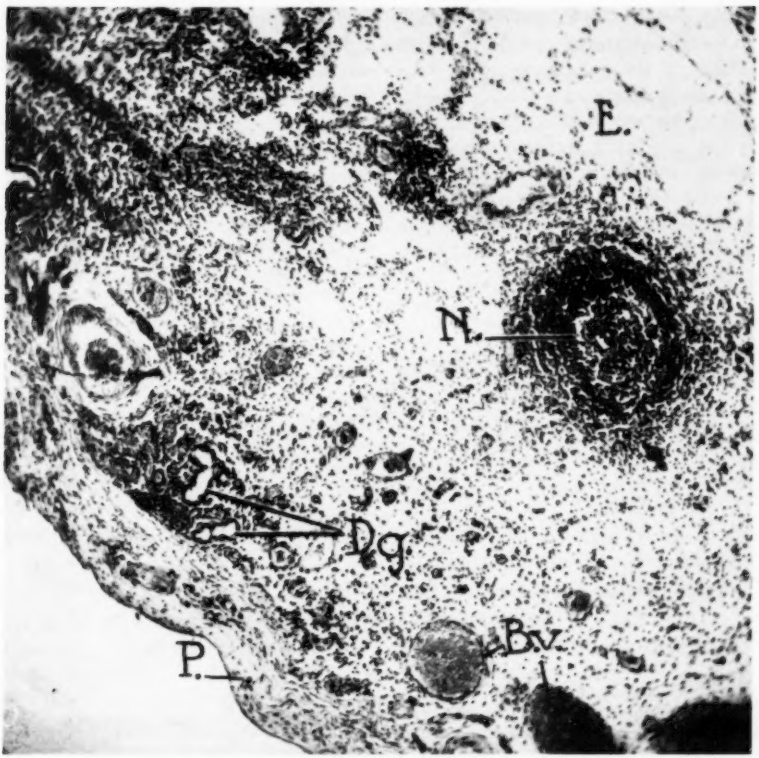

FIG. 12—Chronic purulent sinusitis. E—edematous tunica propria; N—nodule of lymphoid tissue; Bv—congested blood vessels; Dg—deep glandular (periglandular) infiltration; P—periosteum. Note the sharp line of demarcation between the tunica propria and periosteum. The tunica propria is the layer that is most active in inflammation. This girl, age 13 years, had chronic sinus-bronchitis which failed to respond to intranasal sinusotomy. She was cured by exenteration of the infected membranes. The infection is deep seated, as shown in this photograph. No evidence of allergy. See figure 19 (A), which is from the same case.

In short, the tunica propria plays a vital part in the defenses of the body against infection and a high degree of local tissue immunity rests in the mesenchymal elements.

Bacteria, allergens and toxins stimulate the mesenchymal cells and cause them to proliferate and produce not only fibrous connective tissue, but also macrophages and even lymphoid follicles (fig. 12).

Periosteum

The layer of stroma that lies below the tunica propria, because of its general character and location, acts as a periosteum and may resorb or deposit bone, according to the occasion. It is compact in structure and composed of thick collagen fibers, intermingled with a few elastic fibers. Although it is rather rudimentary when compared with the periosteum of other bones and varies considerably from place to place over ridges and spurs, as anyone who has ever performed a submucous resection can testify, it none-the-less is tougher than the tunica propria and more resistant to certain collagen diseases. As a

matter of fact, in nonsuppurative sinusitis the inflammatory reactions often stop short of the periosteum (fig. 12), and in many instances the periosteal layer tends to become tough and fibrous while the overlying tunica propria degenerates into a soft hyperplastic polypoid mass. This fact accounts for the ease with which a subperiosteal exenteration of the antral lining can be accomplished in some, but not all cases.

Intercellular Substance

Having reviewed, layer by layer, the architecture of the connective tissues that enter into the construction of what at first seemed to be a simple membrane, we finally reach the point where we can approach the subject of collagen disease in the nose and sinuses. To clarify our ideas, let us digress for a moment and emphasize the meaning of the somewhat vague term of intercellular substance.

According to Maximow and Bloom:[23]

> There are four main types of connective tissues, all characterized by an abundant intercellular substance: (1) blood and lymph, (2) connective tissue proper, (3) cartilage, and (4) bone. In the blood and lymph the intercellular substance is liquid. In the connective tissue proper, of which there are many types, the intercellular substance always contains fibers and varies from a soft jelly to a tough fibrous mass. In cartilage, the intercellular substance contains masked fibers, and has a rubbery consistency. In bone, the fibrous intercellular substance is impregnated with lime salts.

With the best of histological methods we see only a few intercellular elements and must guess at the rest. The tunica propria of the sinuses, however, contains all of the cellular and intercellular components that characterize "connective tissue proper" throughout the body. Among other things, the fibroblasts manufacture such intercellular products as (1) fibrils, (2) fibers, and (3) an amorphous ground substance.

Collagen fibrils are extremely thin (some are ultramicroscopic) and, in certain organs, the fibroblasts associated with them are called reticular cells.

Collagen fibers are much thicker and form a familiar pattern of pink-staining fibers, which, under the electron microscope, prove to be bundles of fibrils.

Ground (cement) substance binds fibrils, fibers, and cells together, and when liquefied by hyaluronidase, the spreading factor, the tissue spaces become more permeable to fluids.

In perennial nasal allergy, the tissues become boggy and the hydrated cells and fibers assume a myxoid (resembling myxomatous tissue) gelatinous appearance. Does the cement substance, in this case, lose some of its strength, like wet glue? And why does fibrinous inflammation produce the opposite effect — so much dense cicatricial tissue?

In some individuals, the whole mucoperiosteum may be loosely formed, and in others, the structure tends to be more compact. This difference apparently is due to a greater preponderance of fibrils and areolar spaces in one and a greater development of fibers and cement substances in the other (fig. 13). Variations of this sort may be constitutional in origin and may account for the unpredictable course that inflammations follow in different individuals, and sometimes in different parts of the same individual.

> The use of sclerosing and cauterizing agents in the nose, I believe, alters the character of the intercellular substances and provokes deposition of fibrin and cement substances in the tissue spaces, which leads to formation of a more fibrous (scar) tissue and tends to reduce the areolar spaces and swelling. A certain amount of fibrosis is desirable when it occurs in the right places.

Collagen Disease

As used by Klemperer[19] the term "collagen system" refers to fiber form-

ing connective tissues throughout the body. In the so-called collagen diseases, the pathologic changes appear in the intercellular substances, i.e., in the ground substances and in the fibers. As stated, collagenic fibers and/or reticular fibrils are found nearly everywhere and yield the colloidal solution or gelatin that is called collagen. Collagen forms a large part of the body, appearing in many organs, heart valves, blood vessel walls, etc.

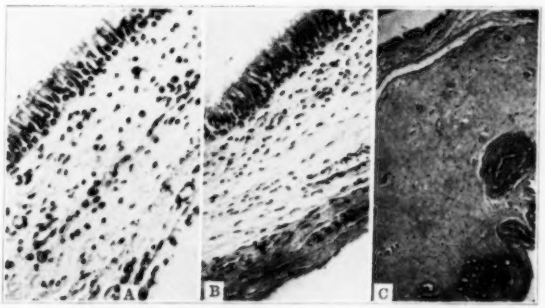

FIG. 13—(A) Entire mucoperiosteum composed of loose fibrillar stroma; (B) typical mucoperiosteum composed of a more fibrous stroma; (C) dense and more fibrous sinus mucoperiosteum. (C) is from a patient who had sinusitis for 25 years. It finally was cured and became asymptomatic. The lining was thick but tough and fibrous. (Semenov[26])

It occurs in the nose and throat, and no one can deny the high incidence of inflammation and allergy in this area of the body. One of the most obvious examples of collagen disease is nasal polyposis. It occurs in the nasopharynx in tonsil and adenoid reticulum, and in laryngeal nodes and polyps. Why do these parts suffer simultaneously in so many of our patients? Is the collagen system the common denominator?

Tissue Fluid and Lymph

The serous cysts (interstitial cysts) that we find in the antrum contain a large amount of amber fluid, which may be tissue fluid or lymph. With histologic methods alone it is difficult to decide which of these two possibilities is the correct one, and at present I think that there might be two types of cysts. One type has thin walls (fig. 1) and the other has thick ones. When the fluid is washed out of the antrum, it coagulates slowly, like lymph. Are some of these cysts congenital lymph sacs?

Tissue fluid does not circulate like blood, but ebbs and flows between the cells, under the influence of a great many factors. Like lymph, it contains salts, albuminous and other materials that are invisible in microscopic slides. It nourishes the tissues and also helps to moisten the surface of the mucous membranes. Lymphatics are pulled open by distention of connective tissue fibers. They drain the tissue fluid away, removing toxins and also colloids. A wide variety of biochemical and physical (e.g., osmotic) disturbances can permit abnormal amounts of fluid to remain in the tissues.

Lymphatic capillaries are almost as extensive as blood capillaries. Their identification in microscopic sections, however, is not easy, and rounded spaces in edematous tissues may or may not be lymphatic channels. The channels are not as well outlined as blood vessels (the latter have thicker walls, fluid under pressure, and contain red blood corpuscles). The lymphatics look empty, contain a few lymphocytes, have thin walls, and when dilated the girth sometimes is very great and difficult to outline.

According to Mullin and Ryder,[29] lymphatics of the sinuses enter and leave only by way of the ostium. It seems logical to assume, therefore, that obstructions in and around the ostium or anywhere along the lymphatic outflow may cause stasis at a distant point (e.g., the lateral wall of the antrum).

The lymphatics of the nasal and sinus mucous membrane are continuous and the main trunks drain into the postnasal area (region of the eustachian tube and pharyngeal recess). The lymphoid tissue situated in the lateral recess, in my opinion, is an important hub and enters into a variety of widespread nasal and aural disorders.[38]

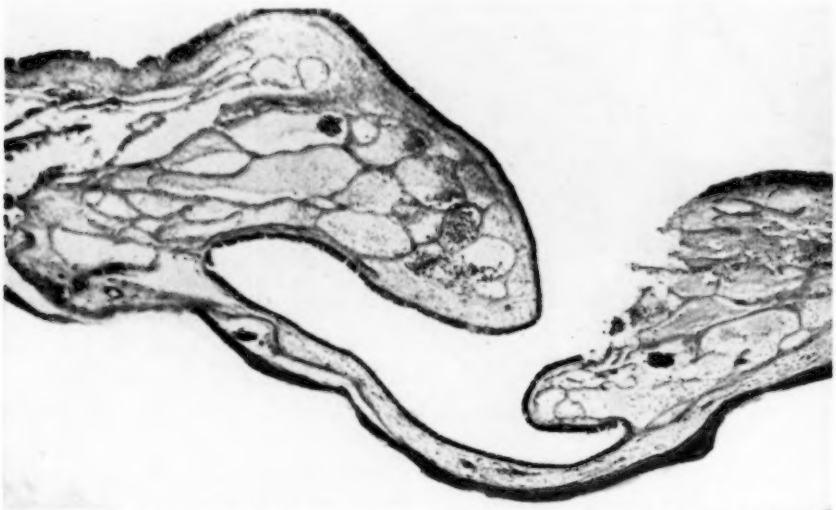

FIG. 14—Ethmoidal sinusitis, allergic. Note the normal mucosa in the middle of the section and the polypoid swellings on either side. This photomicrograph shows how polyps begin. First there is a wheal-like edema that distends the loose areolar stroma, and then the redundant mass begins to sag. The distended fibers are injured and the edema fluid in this specimen is loaded with eosinophils. Compare with figure 3, which is normal and taken at nearly the same magnification (20 X).

Edema

Connective tissues are endowed with tremendous powers to resist injury and repair wounds. As a consequence, they produce new cells, new fibers, and new intercellular substances. Sometimes, however, the fibroblasts may be so young or so edematous that they not only look myxoid (resemble myxomatous tissue), but actually fail to function properly, which is typical of hyperplastic cells in general.

When water accumulates in the tissues, it may appear in a free or combined form, and in an intracellular or extracellular position. The hydrops of the fibroblasts and swelling of the fibers and jelly-like intercellular substance can be seen in typical nasal polyps. In polyps, therefore, the edema is both intracellular and extracellular. The former accounts for the myxoid appearance of the cells and the latter accounts for the distention of the tissue spaces.

Edema is due to many things: (1) inflammation and abnormal permeability of the blood capillaries, (2) venous stasis, (3) lymphatic obstruction, and (4) biochemical (eg., cortisone) disturbances.

The first change in the delicate sinus muco-

periosteum is prolapse of the tunica propria (fig. 14). In lymphatic obstruction (inflammation) of the channels, formation of clots may interfere with the flow of fluid from the tissue spaces and the persistent back pressure, as time goes on, favors the formation of serous cysts.

Mucous membranes, like other tissues and organs, depend upon a constant supply of enzymes, endocrines, food and oxygen; and an equally constant removal of waste products, that is, an adequate blood supply, and circulation of lymph and tissue fluid.

FIG. 15—Arteriole and venule in the tunica propria of a sinus mucous membrane. There is a slight thickening of the wall of the arteriole (arteriofibrosis) and the surrounding stroma is fibrotic. Note the fibrocytes.

Polyps, whether bacterial or allergic in origin, have a poor blood supply and a tremendous amount of edema, which, of course, begins with abnormal capillary permeability, and a wheal-like swelling of the tunica propria. The locus minoris resistentia or anatomical areas of the nose that suffer the most damage have been mentioned. The presence of perennial allergy and/or chronic infection produces profound effects in the collagen and perpetuates the edema. The end result is degeneration, and as has been stated, it is worse in the parts of the nose and sinuses that have the most delicate tunica propria. Mechanical factors (e.g., antronasal polyps or aerosinusitis) aggravate the situation, especially when the stalk of a pendunculated polyp strangulates in the sinus ostium and the circulation becomes impaired. This does not explain all polyps, but it certainly clarifies the persistence of some.

I do not wish to create the impression that they always begin in the sinuses. They do not. Nasal polyps are far more frequent, as anyone who has ever practiced rhinology can testify, but the pathology is easier to explain in the sinus linings because of the simplicity of the histology.

BLOOD VESSELS

The blood supply of the sinuses is simple and follows a textbook pattern; the arteries and veins enter the sinuses through foramina in the surrounding bony walls and rest in the periosteum. From this location they give off branches that divide and subdivide into arterioles and venules, arteriovenous anastomoses (Prinzmetal, et al[31]), and capillary loops. When the arteriovenous shunts are open, the circulation by-passes the capillaries because the shunts have a larger diameter than the capillaries. When they are closed, it detours through the capillaries. The flow of traffic is controlled by an elaborate stop-and-go system—the autonomic system and vasomotor nerves that regulate the arteries and veins, shunts, and capillaries.

When a great deal of congestion and swelling occurs, the capillaries seem to spring upward, almost at right angles.

The veins penetrate the bone indirectly, and according to Bleicher[4] form a plexus on (1) the nasoantral wall (region of the ostium) which joins the veins of the nose, and (2) a lateral (zygomatic plexus) which converges on a vein that leaves through the

FIG. 16—Normal nasal mucous membrane, inferior turbinate. Note the epithelium, clean tunica propria, which is more compact than that of the sinuses and thicker, and the cavernous blood vessels, in the midst of which a cluster of seromucous glands may be seen. The blood has been washed out of the erectile tissue, which is fully dilated.

tuberosity of the antrum and eventually reaches the pterygomaxillary fossa. Veins can transmit infection by thrombophlebitis, as well as the tissue spaces around them (fig. 15).

The nasal mucous membrane is outstanding because of its extreme vascularity and massive deposit of neatly arranged venous channels (erectile tissues) (fig. 16). The walls of these cavernous veins are thick and composed of fibro-elastic tissue and smooth muscle which is developed to a degree ordinarily associated with arteries. They characterize the mucosa of the turbinates and septum, where they rest in the deeper layers of the tunica propria and may be identified histologically by their irregular shape in cross sections, which is unlike the perfectly round one of arteries (fig. 17).

In addition to erectile tissue, the nose contains a great many typical arteries, veins and capillary loops. The circulation between the erectile tissue and ordinary blood supply is governed by an elaborate arrangement of shunts and anastomoses, which is so extremely sensitive that it responds to changes of temperature and emotion (crying).

Some of the blood vessels pass through the ostia of the sinuses and others simply penetrate the intervening walls. The sinuses, however, are utterly devoid of erectile tissues.

Vascular Reactions

The work of Cohnheim (1889) taught us that the process of prime importance in an area of inflammation is the vascular reaction, especially the increased capillary permeability, congestion and slowing of the blood flow, escape of fluid, margination of leukocytes and diapedesis through the capillary walls. The hyperemia associated with inflammation does not occur through a greater flow of blood, but rather through congestion. A similar vascular reaction is observed with histamine, which causes capillary and arteriolar dilatation and constriction of arteries and veins. Although it produces dilatation of the capillaries and an exudation of fluid, Morgan[28] showed that there is no fibrin in the area of reaction, which is true of allergy too. When we phenolize or cauterize the nose, we get a fibrinous reaction which, oddly enough, improves the situation. The histamine response includes:

1. Local dilatation of the capillaries and venules by direct action
2. Widespread dilatation of surrounding arterioles from a local axon reflex
3. Local increased permeability of the capillaries
4. Exudation of fluid (wheal formation)
5. Edema and distention of the tissue spaces

About twenty years ago, I remarked that a bee sting looks like a histamine reaction. Needless to add, anyone who has ever been stung by a bee knows the difference, and I mention this homely fact merely to emphasize the point that appearances are deceiving and everything is not due to histamine.

Hyperemia, local anoxia and escape of fluid and plasma proteins into the tissue spaces is governed by a combination of factors.

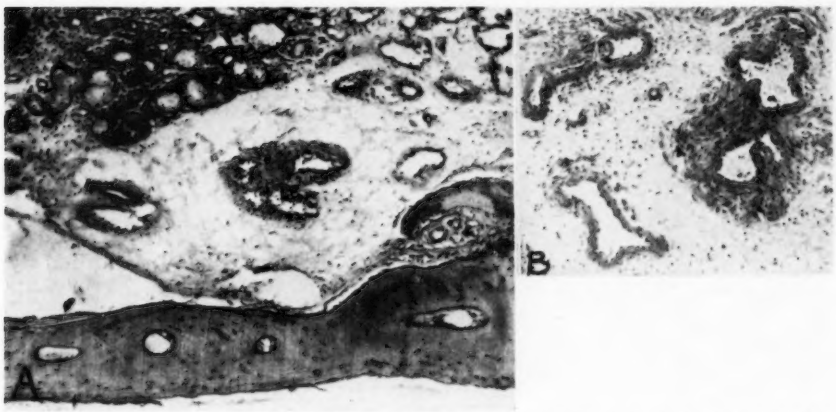

FIG. 17—(A) Mucoperiosteum of the inferior turbinate showing seromucous glands in the upper left-hand corner, erectile tissue in the center of the field, and the line of cleavage from the bone, which is uneven and hooks into the membrane on the right. This makes a submucous resection of the inferior turbinate more difficult than one of the septum. (B) Erectile tissue under higher magnification showing the irregular shape of these blood vessels when they are contracted. This is the way that they usually look in surgical specimens. Some chronic rhinitis is present.

GLANDS

The nasal mucosa is endowed with a rich and abundant supply of mucous glands. Their size and number are directly proportioned to the thickness of the tunica propria and to the vascular supply (fig. 16). The large ones are of the branched tuboalveolar type, and some alveoli are serous, while others are mu-

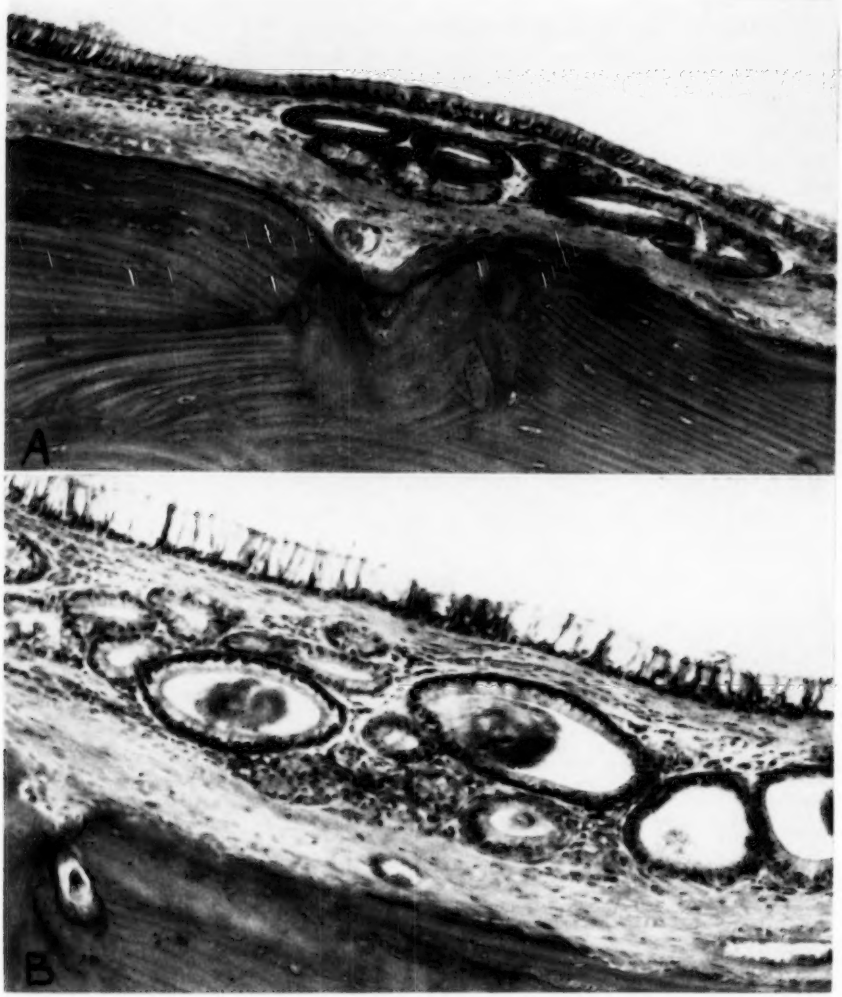

FIG. 18—Glands of the maxillary antrum. In (A) the glands are nearly normal, and in (B) they are slightly dilated. A mild degree of inflammation is present in (B) as evidenced by the increased number of goblet cells and scattering of round cells (lymphocytes) in the tunica propria. Note the vein lying in an osseous groove in (A). Little or no edema is present in these sections. (AFIP neg. 82200 x 250).

cous in type. Either one of these types is capable of elaborating mucus when stimulated. Glands are very numerous over the anterior parts of the inferior and middle turbinates and are less numerous in the meatuses, especially the superior meatus. They are supplied with nerve endings which regulate their activity and secretion. When hyperactive, the alveoli become bulky and voluminous, and contain a preponderance of mucous elements.

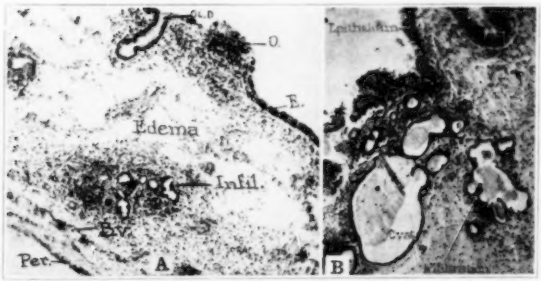

FIG. 19—(A) Chronic purulent maxillary sinusitis showing edema and perglandular infiltration. Gl D—gland duct near which the epithelium (E) is breaking down into an ulcer (O). B—dilatation of glandular acini and rupture of one cyst into another produces a large retention cyst. See figure 20, which is a gross photograph of the same specimen. (Semenov[88])

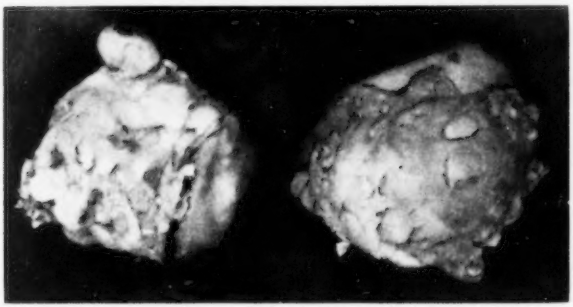

FIG. 20—Chronic maxillary sinusitis, cystic, mixed allergy and infection. At the time of operation both antrums were filled with pus and the membranes were studded with multiple cysts which exuded mucus and mucopus when opened. Hyperplasia and thickening measured 4 mm. Histologically (figure 19—B) there was a marked edema of the stroma and dense infiltration of leukocytes, many eosinophils and polymorphonuclear cells. The epithelium was hyperplastic and undergoing mucoid degeneration. The basement membrane was thick and hyalinized. Lymphatic follicles were also present in the stroma. The glands were cystic and in all stages of distention, producing the above picture. This case illustrates a combination of allergy and infection. The allergy leads to bilateral pathology.

In the *paranasal sinuses*, the glands tend to be small (fig. 18) and few in number. The clusters lie in the tunica propria and thus have a poor support. The flimsy areolar stroma is no match for the pressures that can build up within them. Almost anything that chokes the ducts, the slightest swelling or infiltration of the tissues, can lead to cystic changes (fig. 19). In fact, the number of mucous retention cysts encountered in the sinuses is great and far exceeds the incidence in the nose (fig. 20).

The ethmoidal, sphenoidal and frontal sinuses contain some glands in the neighborhood of their ostia, but they are few in number and in many instances vast areas of mucous membrane are utterly devoid of them (fig. 3). In the maxillary antrum, on the other hand, a number of glands appear in the medial half of the antrum and sometimes extend laterally as far as the molar region, where they finally disappear. A relatively large gland, which often becomes cystic, is found in the anterior

part of the nasoantral wall. Now and then the excretory ducts appear to spring from an invagination of the surface epithelium, and an occasional cluster of glands may be interposed between the periosteum and the bone. As stated, glands are controlled by nerve impulses. Goblet cells, on the other hand, operate in an entirely different manner and cannot be controlled with atropine.

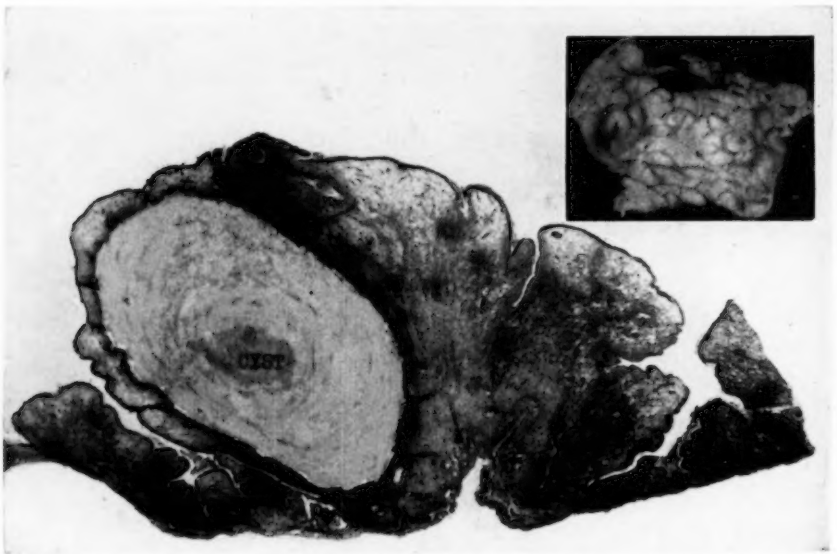

FIG. 21—Large mucous retention cyst in chronic maxillary sinusitis due to a combination of allergy and infection. (10X) A view of the gross specimen is shown in the upper right hand corner. Both maxillary sinuses were involved and the pathologic condition was identical on both sides. The patient had hay fever and severe asthma.

EPITHELIUM

The pseudostratified ciliated columnar epithelium that is found throughout the respiratory tract (the nose, sinuses, eustachian tube, trachea, larynx and main bronchi) is so similar in appearance everywhere that many pathologists call it "respiratory epithelium." Although only a single layer actually is present, it is made up of four types of cells, each of which represents a step in the process of maturation:

1. *The Basal Cells* are cuboidal in type and have round, comparatively large, dark-staining nuclei that line up along the basement membrane. These are the young cells that regenerate the epithelium.
2. *The Intermediate (Transitional) Cells* are more or less cylindrical and probably represent the transition to a more mature generation of cells.
3. *The Ciliated Columnar Cells* are full grown and taller than either of the preceding. The nuclei are oval and form an irregular row above those of the basal cells. The cilia are from 5 to 12 micra in length, and each cell supports 18 or 19 cilia.
4. *The Mucous (Goblet) Cells* may or may not have a "goblet" shape but they always contain mucus. They probably are modified ciliated columnar cells, and now and then a few cilia still cling to them. With mucicarmine the mucus stains red and with hematoxylin it takes a very pale bluish stain. Mucous cells may appear singly or in patches, and are most abundant in catarrhal inflammation.

The epithelium of the sinuses measures 25 to 40 micra in height, being slightly taller in the antrum than it is in the other sinuses. Under normal conditions it is completely covered with cilia, and the cilia always propel mucus toward the natural ostium, even when we create a large antrum window and establish dependent drainage.

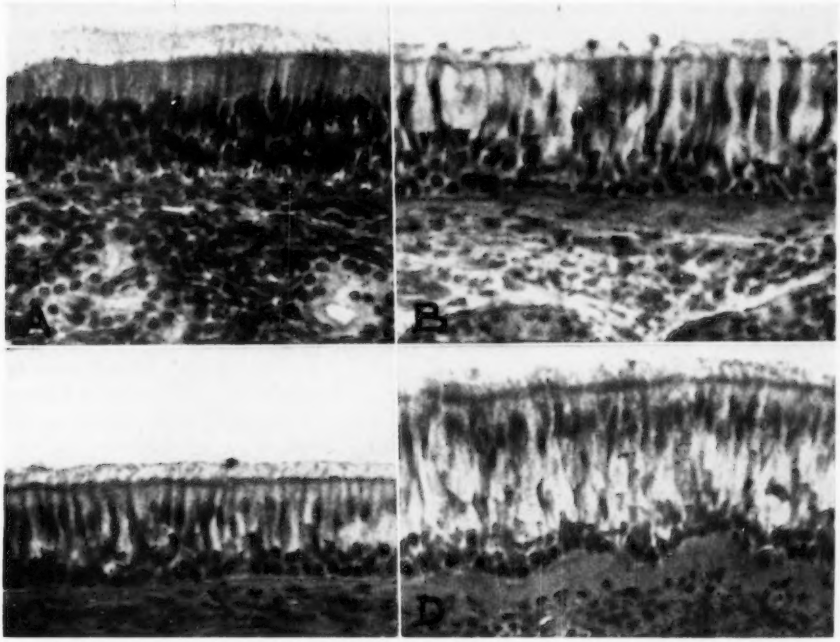

FIG. 22—(A) Olfactory epithelium (note nuclear zone and absence of basement membrane); (B) epithelium of nasal septum; (C) pseudostratified ciliated columnar epithelium of ethmoidal sinuses; (D) inferior turbinate (note basement membrane and greater height of epithelium). Magnification 500 X.

The epithelium of the nasal mucous membrane, in keeping with its more strenuous duties, is 35 to 74 micra thick and is ciliated in all parts of the nose, except in the preturbinal region, and in the olfactory region.

The cilia propel mucus toward the pharynx and are capable of cleansing the nasal passages within half an hour. As stated above, they are deficient in the anterior part of the nose, owing presumably to dessication and physiologic metaplasia. The epithelium in this location, therefore, is more or less stratified, and, as shown by Hilding,[14] the cilia virtually dry up with excessive ventilation. Areas of stratified or transitional epithelium are natural over the anterior tips of the turbinates and parts of the septum facing the inhaled (dry) air, but are unnatural within the meatuses (under the turbinates) where, according to Proetz,[32] the exhaled (moist) air flows on its way outward.

Olfactory epithelium: The olfactory area occupies the upper third of the nose. It has a special function (olfactation), and lacks (1) ciliated cells, (2) a basement membrane, (3) erectile tissue, and (4) mucous glands.

The epithelium is composed of olfactory cells, low-lying basal cells and taller sustentacular cells. The olfactory cells are highly

specialized and cannot regenerate once they are injured. The tunica propria contains lymphoid tissue and special serous glands, known as Bowman's glands, which are essential to olfactation. The olfactory (sensory) cells are fusiform bipolar neurons and have hairlike nerve endings. The surface of the epithelium is covered by a semifluid cuticle which contains the olfactory hairs (dendrites). The spherical nuclei of the olfactory and sustentacular cells are packed into the basal part of the epithelium (fig. 22).

NASOPHARYNGEAL PITUITARY GLAND

In addition to olfactory epithelium, another highly specialized epithelial structure occurs in the *pituitary mucous membrane* and might be mentioned at this time because of its unexplored clinical importance in rhinology. Let us take a postnasal mirror and try to understand what is going on right under our very nose. The word pituitary is onomatopoeic or imitative in origin. It comes from the Latin, pituita, meaning phlegm. As a matter of fact, the nasal mucous membrane was rightly called the pituitary membrane by our predecessors and, no matter how you look at it, the name still fits—and for several good reasons.

A small accessory deposit of pituitary gland tissue can be found in at least 95 per cent of persons. It is located in the sphenoidal region and lies just posterior to the vomerine junction, i.e., in the midline. It is composed of cells exactly like those of the anterior lobe of the pituitary gland and is called the pharyngeal pituitary gland. Like other accessory or aberrant endocrine tissues, it marks the trail of the main organ. The anterior lobe of the pituitary gland begins as an epithelial evagination known as Rathke's pouch. From this anlage, epithelial cells pass through the area that later becomes the body of the sphenoid bone and come to rest in the region of the sella turcica. Some pituitary tissue remains in the nasopharyngeal area throughout life and marks the anlage.

Melchionna and Moore[25] found a pharyngeal pituitary gland in fifty-one of the fifty-four cadavers that they examined, and describe it as a well-circumscribed structure. The largest one that they found measured 6.62 mm. in length and 1.15 mm. in width and 0.35 mm. in thickness. In the cases that had well-differentiated cells, the histology of the pharyngeal tissue looked exactly like the anterior lobe of the pituitary gland. In other cases, differences were encountered in the relative numbers of acidophilic and basophilic cells. In 25 per cent of the cases there were no chromophilic cells and in 35 per cent the acidophilic and basophilic cells were absent. In a few cases they found definite vacuolization of the cells, which is a sign of function and secretory activity. They remarked that "under normal conditions of growth and activity it is unlikely that the pharyngeal pituitary gland can produce any significant physiological function, but in some cases of altered structure or activity of the pituitary gland, it cannot be denied that the pharyngeal pituitary gland may undergo structural alterations and serve as an endocrine organ." They cite Christeller and one of their own cases in which there were conspicuous eosinophilic cells in the pharyngeal pituitary associated with Froelich's syndrome and teratoma of the testis, respectively, indicating possible physiologic activity. They concluded that certain cells in the pharyngeal pituitary are histologically identical with those in the anterior lobe of the pituitary gland, and, when cells of this type occurred, they found histologic evidence of function, as shown by vacuolization of the cells.

The influence of the pituitary gland on the entire body is so profound that we have only just begun to observe the impact. The somatotropic hormones (STH) and the adrenocorticotropic hormone (ACTH) are so potent, that a few molecules in the right place can produce profound effects. We know so little about the pharyngeal pituitary, aside from the fact that it occurs in nearly everyone, which serves to indicate that it is a normal rather than abnormal condition, that one might rightfully ask: Does it take any

part in nasal allergy? Is it related to atrophic rhinitis? Is there any possible connection with the autonomic nervous supply of the erectile tissue and the sphenopalatine ganglion, the only ganglion that lies fairly close to the surface of the body?

Inflammation and Epithelial Catarrh

With the slightest sort of irritation there is an increase in the number of goblet cells; and for every goblet cell that appears, one ciliated cell disappears. As the reaction becomes more intense and more and more mucus-forming goblet cells are developed, the epithelium begins to change its tactics as well as its appearance, assuming the characteristics of "catarrhal epithelium" (fig. 23).

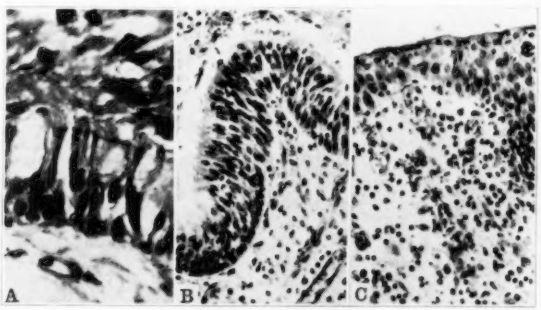

FIG. 23—(A) catarrhal epithelium (note the typical goblet cell and nearby ciliated columnar cells). The mucus on the surface is loaded with desquamated cells. (B) epithelial hypertrophy in purulent sinusitis. (D) metaplasia and loss of cilia in the same condition as "B." All of these changes are reversible. (Semenov[36])

When a great many cilia are damaged or destroyed, ciliary insufficiency arises; and when vast numbers of cilia disappear and are replaced by "goblet" cells, overloading of those that remain behind certainly ensues. In catarrhal inflammation the strategy of the epithelium seems to be one of mobile warfare, hit and run. As a consequence, the surface epithelium hurls mucus at the enemy, sloughs away and is reduced to basal cells, which regenerate more troops. The discharge becomes loaded with dead and dying "mucous corpuscles," a handy term coined years ago to describe the cells that occur in mucus. In the heavy flow of mucus that stems from catarrhal epithelium we find much epithelial debris and many leukocytes.

With prolonged irritation or more intense irritation, the strategy changes to one of defense in depth. The desquamation and constant repair and regrowth of new epithelium leads to stratification, which combines catarrhal and hyperplastic processes in varying degrees, depending upon the stage of the war. When many mucous cells are present, the inflammation is still in the acute or active stage.

In allergy, therefore, the perennial catarrh may eventually lead to epithelial hyperplasia and even metaplasia.

In chronic purulent sinusitis, the picture is more or less circumscribed and slightly different; some areas ulcerate or disintegrate, and others undergo hypertrophy and hyperplasia, sometimes metaplasia, as shown in figure 23. As a rule, the battle is not of the goblet cell hit-and-run type, but rather hold-that-line and defense-in-depth. Metaplasia, of course, is a sign of severe inflammation, and with it comes a loss of cilia and cuboidal cell stratification of the epithelium. Goblet cells come in when the

FIG. 24—Chronic ethmoiditis with metaplasia and transformation of the epithelium into unciliated multilayered cuboidal cells. (Kistner and Semenov[16])

fight is won and mop up the scene. Complete metaplasia to stratified squamous epithelium, with prickle cell formation, is encountered when all other strategy fails and probably is irreversible.

Mucous membranes elaborate mucus for much the same reason that skin elaborates keratin; namely, self protection. Although both of these products serve useful purposes, an excess of one or the other is undesirable, and chronic catarrh, like scaliness of the skin, is pathological.

MUCUS

In dealing with the autonomic nervous system, the blood vascular system, the endocrine system, the macrophage system, the collagen system, infection, inflammation, and allergy, we encounter

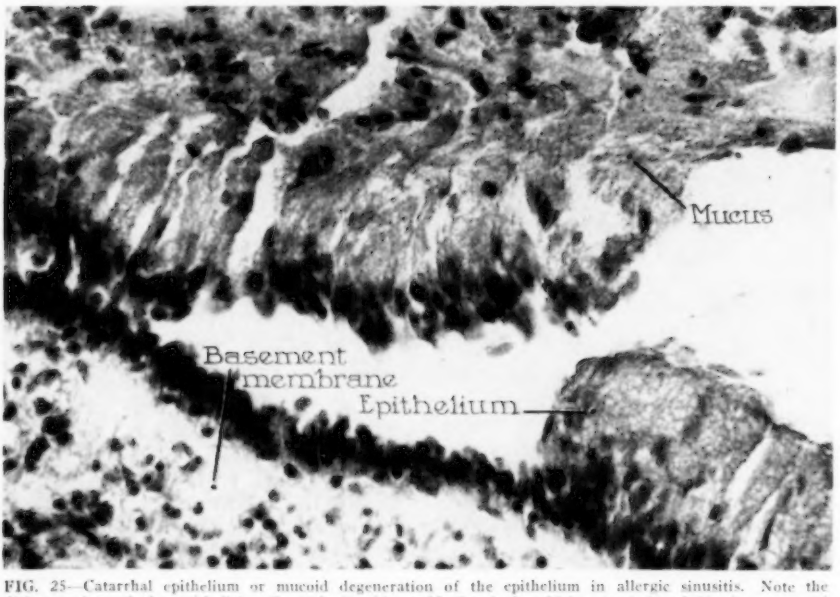

FIG. 25—Catarrhal epithelium or mucoid degeneration of the epithelium in allergic sinusitis. Note the disintegration of the epithelial cells and sloughing. Much of the thick mucus found in the nose and sinuses is formed in this manner. There are no more ciliated cells in this area. They have been converted into mucous cells. When these cells desquamate, only a remnant of basal cells will remain behind. Iodides apparently help liquefy this type of mucus.

such powerful forces arrayed in almost endless combat formations that no one will disagree when I say there is more to a mucous membrane than its cilia. With this in mind, let us put these petty janitors in their place, and not confuse moving pictures of normal tissues with the devastation that is wrought when a storm strikes and the silly cilia dwindle to insignificance, in fact run away and hide until it is all over (fig. 25).

According to Lucas[22] and Proetz,[32] mucus normally is deposited in two layers, a viscid top layer that catches particles of dust, pollen and bacteria, and a thin, watery layer that lubricates the cilia. As long as mucus of the right sort is present and as long as the cilia whip normally, it is able to move freely. But when very thick or very thin material is formed, it may fail to move even though the cilia beat vigorously (fig. 26).

As stated previously, a good picture is worth a thousand words. To understand the cilia and mucus film, one should see the motion pictures made by Arthur Proetz and, more recently, by G. Edward Tremble. These films show cilia at their best, like movie queens.

As regards degeneration of the cilia and epithelium, some pathologists object to the term mucoid degeneration, contending that the term mucinous degeneration is more appropriate. Whether this is so or not, the limpid normal secretion that is derived mainly from glands is clear and viscid, and the abnormal material that comes in part from glands and in part from the disintegration and desquamation of surface epithelium is thick and turbid. The ancients said "pitui," and spit it out because phlegm is acrid and irritating, and anything but soothing. That is why it feels so good to have it washed out of the nose—cilia or no cilia.

BONE AND PNEUMATIZATION

Bone, it has been said, is not as solid as it looks—some bones are filled with marrow and others are hollowed out and filled with air. The paranasal sinuses are examples of the latter condition, and the process by which they are formed is known as "pneumatization." During pneumatization, which begins in embryonic life, the air cells follow in the wake of marrow spaces.

The embryonic mucoperiosteum of the newly formed cavity is very thick and the periostal aspect is uneven, that is, it indents the bone. As pneumatization proceeds and the air cavity grows in size, the mucoperiosteum becomes much thinner. The glands tend to remain behind in the paleosinus; and as the mucosa stretches into the neo-sinus, they finally disappear. This explains the difference in thickness between the nasoantral wall and lateral wall of the antrum (table I), and certain peculiarities of the frontal sinus, which may be regarded as a neo-sinus because it develops about the age of six or seven years, long after the others.

There are 25,000,000 cases of rhinitis during the season, and hardly a person reaches maturity without an attack of complicating sinusitis. In allergic children the attacks are sometimes continuous, but pneumatization goes on without signs of interruption in most cases, contrary to Wittmaack's theory of arrested pneumatization. As we all know, the pituitary gland has something to say about bone and pneumatization and can cause acromegalia. Why stop with Wittmaack's obsolete theory?

APPLIED PATHOLOGY

Applied pathology is practical pathology. It can be summed up in two words—"say, Ah!" Every time we utter this battle cry, a whole lifetime of study, experience and applied pathology snaps into action.

It might seem easy, to the uninitiated, to differentiate one form of rhinitis from another, but the task is not an easy one. The slightest familiarity with the problems of rhinology will reveal the difficulties and almost constant mixup in diagnosis, e.g., overlapping of sinus infection, nasal allergy and/or neurosis. Much of this difficulty is due to "the great imitator," but not all of it.

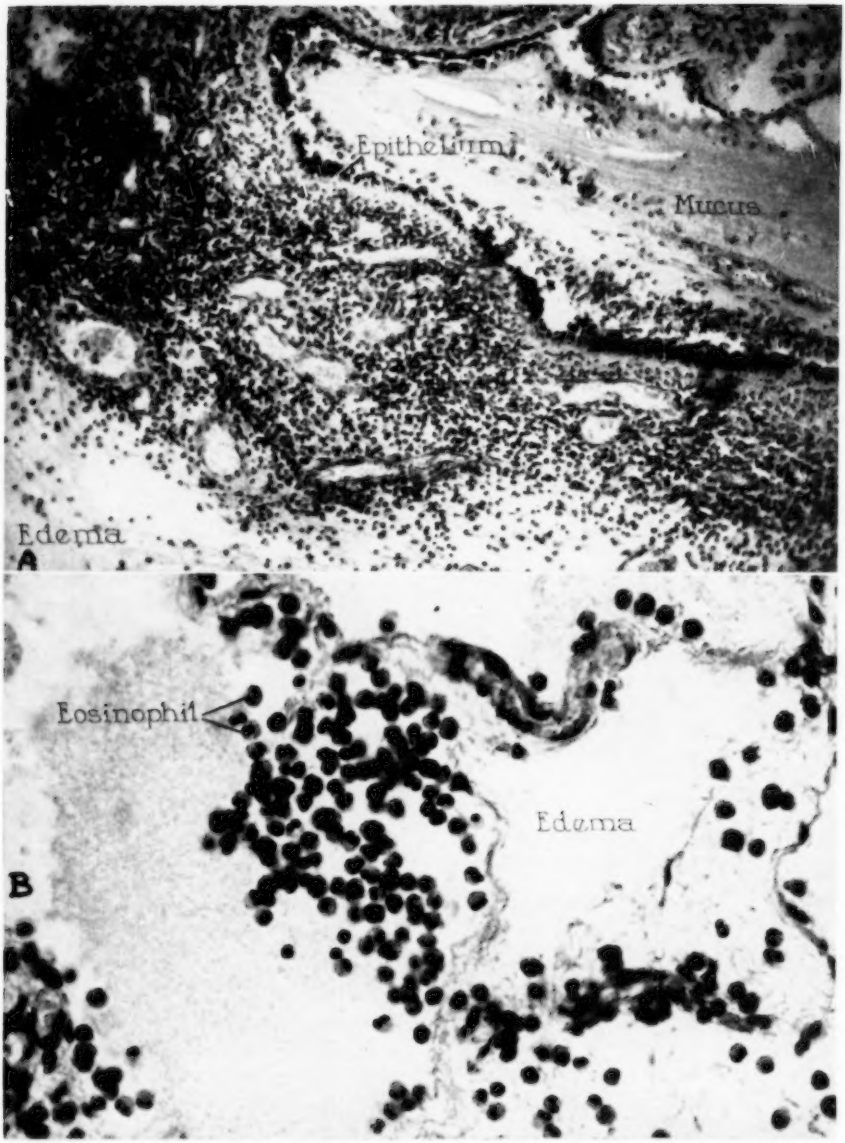

FIG. 26—(A) Allergic sinusitis showing catarrh and mucoid degeneration of the epithelium. Note the complete loss of epithelial continuity and flow of mucus from the surface. This is not an artefact. The underlying basement membrane is hyalinized and the subepithelial stroma heavily infiltrated with eosinophils. (B) Higher magnification of the edematous tunica propria showing the eosinophils and round cells. Note the tissue space and fibers.

FIG. 27—Nasal polyps (allergic) protruding from both nostrils of a boy.

As regards the clinical diagnosis, the healthy nose produces a thin film of clear mucus, which keeps it clean. When an excessive amount is formed, we suspect the existence of a catarrhal inflammation, which may be due to either allergy or infection. When the discharge becomes turbid, or assumes a yellowish or greenish color, infection of the sinuses is suspected. A mere inspection of the nose and throat may or may not reveal the presence of exudate, and in my office stained smears, roentgenograms and, when indicated, skin tests and other diagnostic aids are employed almost routinely.

Practical Classification of Pathology

For all practical purposes we must separate sinusitis into two main types, (1) suppurative sinusitis, and (2) nonsuppurative sinusitis. The first condition invariably is due to infection, and, like pyogenic inflammation anywhere in the body, it responds to a surgical principle that is 200 years old—"*ubi pus, ibi evacua.*" In the second condition, we have an entirely different situation, and in many instances a different etiologic agent or combination of agents.

The time-honored notion that all chronic inflammation begins as an acute infection is a half-truth. Some cases may begin that way, but the vast majority, especially those associated with perennial allergy, are chronic from the very onset, and throughout life they have remissions and exacerbations. In the imperfect state of our knowledge we simply divide the pathology into three stages:

FIG. 28—Hyperplastic (polypoid) maxillary sinusitis. Note the so-called "cobblestone" appearance. This is a typical example of chronic perennial allergy and degeneration of the sinus mucous membrane.

Stage One—inflammatory edema and exudation

Stage Two—thickening and hyperplasia

Stage Three — degenerative changes, polyps and cysts

When we classify sinusitis according to its cause and etiology, we have a type that is due solely to infection, another type that is due solely to allergy, and a third and most devastating type that is due to an overlapping of allergy and infection.

The first type (pure nasal or sinus infection) is by far the most common, and like any other acute infection, it often responds to antibiotics and generally requires adequate drainage. The old saw, "once a sinus, always a sinus," does not apply to these cases.

The second or allergic type tends to be chronic and may be dubbed "once an allergic, always an allergic." Much of the so-called chronic hyperplastic rhinitis and sinusitis is of this type, and in 70 per cent of the cases I found histologic evidence of secondary infection, which brings us to the third type of pathology (fig. 29).

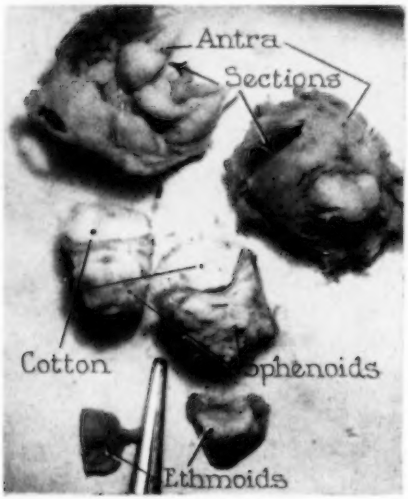

FIG. 29—Pansinusitis. Allergy is a constitutional disease and often affects many parts of the body at the same time. In the nose and sinuses the pathology usually is bilateral as shown in the above photograph of the polypoid tissue removed from one patient. Both maxillary sinuses, both sphenoidal sinuses and all of the ethmoidal sinuses were involved. Infection complicated the problem and pus was found in all of the sinuses. The antral membranes are 1.5 cm. thick, or nearly a hundred times thicker than normal. A bilateral transantral-ethmosphenoidectomy was performed in this case. Heroic surgery of this sort seldom is necessary today.

Bacteriology

There is no specific etiologic agent for all rhinitis or all sinusitis and any one of a great many different pathogenic viruses and/or bacteria can invade the nose and sinuses.

A number of investigators proved that normal sinuses are bacteriologically sterile. Mixtures of bacteria are common in chronic sinusitis, and in four out of five cases, Kistner[17,18] and Semenov[35] found two or three different kinds of bacteria occurring at once, i.e., mixtures of streptococci, staphylococci, Micrococcus catarrhalis, pneumococci, Friedlander's bacilli, influenza bacilli, colon bacilli, diphtheroids, etc. A pure culture was found in only 20 per cent of cases. In suppurative sinusitis the beta hemolytic streptococcus and staphylococcus were the most frequent offenders. In suppurative allergic sinusitis, hemolytic streptococci were recovered in 85 per cent, but in nonsuppurative allergic sinusitis they were found in only 60 per cent. Streptococci of one type or another were found in 94.5 per cent of all of our material, and staphylococci in 70 per cent. These were the predominating bacteria in nearly all of our cultures.

Acute Rhinitis

In textbooks, acute rhinitis is cited as a classical example of catarrhal inflammation. Epithelial damage appears very early, along with glandular hypersecretion and a profuse flow of mucus. The desquamation of cells, singly and in groups, is a prominent feature of the pathology.

Owing presumably to the virus etiology, the leukocytic infiltration of the tissues often is of the round-cell type. But the nasal discharge eventually becomes loaded with polymorphonuclear neutrophils (pus cells)—a point that differentiates acute infection from hay fever. A great many bacteria soon appear in the discharge but they seem to be secondary invaders or presumably chronic inhabitants that flare up during the storm.

Acute Sinusitis

In acute infections we find hyperemia, edema, leukocytic exudation, and sometimes petechial hemorrhage into the tissues, depending on the etiology and severity of the inflammation.

The lesions may be circumscribed or diffuse, but there is no hyperplasia in the beginning. During the stage of congestion and exudation (L., *to sweat out*), the mucosa not only becomes greatly swollen and filled with fluid but may completely obliterate the cavity of the sinus, particularly the frontal sinus, where the walls are in such close proximity that retention and lack of drainage may arise in different parts of the same cavity. During the early stages, the disease is confined to the superficial layers of the tunica propria; and in more advanced stages it may penetrate the periosteum and bone. The spread of infection through the stroma is facilitated by the spreading factor that certain streptococci elaborate and, at times, by lymphatic and venous routes.

As stated above, many kinds of microorganisms are involved and several stages occur, and in the earliest stage large amounts of fluid escape into the tissue spaces. In the sinuses, the resulting edema is unusually great, owing to the areolar structure of the tunica propria. Edema is the outstanding histologic feature (fig. 30), and the water itself will cast a radiographic shadow. If the infection is overcome, the edema subsides with minimal fibrotic changes; if not, the inflammation will go on into suppurative and gangrenous stages. The cellular infiltration consists mainly of round cells and polymorphonuclear leukocytes, which, as a rule, flock to the subepithelial region, but may be scattered throughout the tunica propria.

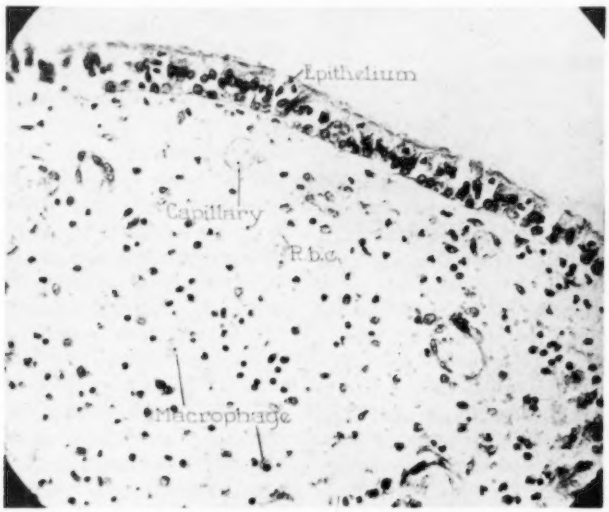

FIG. 30.—Acute sinusitis. Note the edema and dilated capillaries. The leukocytic infiltrate is more or less scattered. (Semenov[31])

Aerosinusitis

In aerosinusitis a negative air pressure (vacuum) develops in the sinuses, owing to poor ventilation and obstruction. The resulting barotrauma causes the mucous membrane to become more edematous than usual and hemorrhagic capillary changes often set in. One may observe the red blood cells here and there in the tissue spaces, along with a scattering of inflammatory leukocytes. The maxillary and frontal sinuses are

more susceptible to this complication, especially during flight and descent from a high altitude — which literally creates a "vacuum sinusitis." The mucous membrane may actually prolapse through the ostium and appear in the nasal cavity as a polypoid, reddish, oozing mass. The only symptom of the sinusitis may be an unexplained hemoptysis. The herniated mucosa can be amputated and the histology examined, if one so desires. Aerosinusitis can arise from either allergic or infectious processes; in either case, the pathology will reflect the underlying disease.

ALLERGY

Allergic diseases have only been recognized as such during the last 40 years. Before that they were uncorrelated and regarded as totally different entities, without any possible connection. It is only in the last 25 years that we have learned about all of the nasal and sinus manifestations. In 1927 Finck[9] called our attention to the eosinophils, and in 1928 Kistner and Semenov[16] were able to confirm the fact that they occur in large numbers in allergic sinuses. During the past 25 years, the work of Hansel[12] has been outstanding in this connection, especially the stain that he has perfected.

Hay Fever

Conditions that to the unaided eye look so much alike may look entirely different when examined under the microscope, e.g., hay fever, coryza, and vasomotor rhinitis. As we look through the microscope, we become impressed with the most colorful one of them all; namely, the tissue eosinophilia of hay fever. Although the leukocytic infiltration may consist largely of lymphocytes and plasma cells, the eosinophils are so gaudy and emigrate through the epithelium so freely that they soon steal the show. This is the combination: eosinophilic infiltration, wheal-like edema, and capillary cyanosis; and a pale, waterlogged, bluish tinge of the mucous membranes.

Vasomotor rhinitis. Vasomotor rhinitis should not be confused with allergy. When biopsy specimens of this condition are examined under the microscope they will be found to be perfectly normal in appearance. The histology, therefore, can be called neither allergy nor infection. In some instances the cause is emotional, which is not at all surprising in view of the similarity of erectile tissue in the nose to that of sexual organs. It is not unreasonable to suggest, therefore, that we call a spade, a spade. After all, the term vasomotor rhinitis simply means that we have an erection of the erectile tissues. The nasal blockage can be differentiated from nasal allergy by a simple cytologic test. The etiology includes rage and anger, weeping and crying, emotional and sexual activities, coldness, dampness and rebound congestion from drugs, autonomic nervous system disturbances, and lesions of the cervical sympathetic trunk. Millonig and his colleagues[27] studied 27 cases in which the *sympathetic* supply was interrupted surgically. They found nasal stuffiness, swelling and increased secretion, and a loss of vasoconstrictor tone, due to uninhibited parasympathetic activity. When the *parasympathetic* nerve supply to the nasal mucous membrane was interrupted, the opposite effects took place; that is, the mucous membranes became shrunken and dry. Walsh[43] believes that the term "vasomotor rhinitis" is a good one, but care should be taken to use it properly. Among the causes, he mentions endocrine dysfunction—thyroid, ovarian and testicular.

Perennial Nasal and Sinus Allergy

In perennial nasal and sinus allergy, as in no other condition, the wear and tear goes on for years and the end re-

sult often is a condition called polypoid degeneration of the nose and sinuses.

There can be little doubt that the chronic edema is harmful, and that it produces not only a great deal of swelling and distention of the soft tissues but also some softening of the adjacent bones. The change that takes place in the middle turbinate, for example, is well known, and half a century back a heated debate took place on the subject of "necrosing ethmoiditis." The controversy eventually subsided, and as we look back upon it now, we realize that allergy was at the root of it. The term "ethmoiditis" unfortunately is still used rather loosely in speaking of this part of the nose.

The striking edema that characterizes perennial nasal and sinus allergy has been called "hydrops mucosae."

Shock tissues. Nature provides us with natural resistance and a host of defense reactions (Perla and Marmorston[30]) but when an allergic individual sneezes and sneezes, his defense reactions try to get rid of something that is not even harmful, e.g., food. His nose runs and runs trying to wash away something that is not even there, and then it erects a barrier so that not even air can enter. When he has asthma, his bronchial tubes contract and he practically suffocates. His defenses certainly are derailed.

The body attempts to rid itself of spirochetes—fifteen years later, the tertiary lesions practically get rid of the body. This is typical of bacterial allergy, (1) infection, and (2) altered reactions that are worse than the bacteria. Lethal granuloma, according to Williams,[45] presumably falls into this category.

Shock tissues are not all alike, but even in a test tube they are capable of reacting in a rapid and intense way against any antigen toward which they may have developed the hypersensitive state.

Allergy	*Shock Tissue*	*Reaction*
Eczema	Epidermis	Within 24 hours after contact
Urticaria	Vascular	Within 15 minutes
Tuberculin	Cutis Vera	Within 48 hours
Hay fever	Vascular	Within 15 minutes
Allergic polyps	Tunica Propria	Within 48 hours

Macroscopically, the tissues assume a pale, grayish color and have a more or less nodular "cobble-stone" appearance (fig. 28). Histologically, the swellings resemble the wheals that appear in skin tests, and in addition to escape of fluid into the tissue, they often display exudation of eosinophils and lymphocytes (fig. 14). Nasal allergy and infection are so exceedingly common that it is only natural that we should be burdened by overlapping conditions.

Although the existing terminology is rich and colorful, there is no point in coining new names—they only confuse us. We all know what hay fever means, even though there is no fever and sometimes no hay. The chief desideratum, therefore, is not a new name but a better understanding of the pathology behind the old one.

The antigen-antibody reaction of allergy involves a combination of three things:

1. The shock tissues—e.g., a ciliated cell.
2. A specific antibody (reagin)—the real trouble maker.
3. The antigen (allergen)—e.g., pollen.

In the hypersensitive state, a certain amount of reagin (antibody) adheres to the cell. When some pollen (antigen) comes along, the antibody and antigen lock horns and the epithelial cell is irritated or injured. When it blows up with mucus, it is trying to wash the irritants away. And so it is with other cells, each in its own way trying to dilute the toxins and lick its wounds.

In the past 15 years it has become apparent that to be of clinical significance, the allergen must be encountered nearly every day, e.g., house dust; that many allergic disorders lack skin positive reactions, no matter how many tests are applied; and that any substance can

combine with a protein, thereby producing a complex molecule that is capable of leading to hypersensitization. Within recent years there has been a more general acceptance of the view that acquired hypersensitivity may enter into the etiology of rheumatic fever, periarteritis nodosa, lupus erythematosus and a number of other diseases that follow infection and an antigen-antibody combination in the tissues.

A wide variety of symptoms and disorders flare up with pharyngitis, tonsillitis and sinusitis; and in cases which the old timers spoke of as "the exudative diathesis, the gouty or lymphatic type," boggy nasal membranes, polyps, and asthmatic bronchitis show up with every "cold." More than a few of these patients have rheumatic diseases, iritis and arthritis, and with every acute infection, particularly the streptococci ones, they have recrudescences in remote parts of the body. What is the common denominator in all of these diseases? Is the collagen system, the shock tissue and hypersensitiveness the explanation? The histopathology of many of these diseases is more or less similar, but not identical, and need not be of the wheal, eosinophil and hay fever type.

The common organisms (streptococcus, staphylococcus, Mycobacterium tuberculosis, Treponema pallidum, etc.) seem to be at the root of most of the evil, and any one of them can cause a disease picture almost identical with the others, because bacterial allergy is "the great imitator."

Bacterial Allergy

A good many years ago, when I was working with Jones and Kistner,[15] I took cultures from the sinuses and studied their virulence in rabbits. With the first injection, inflammatory lesions were produced in the brain, heart, liver, kidneys, arteries and articular joints. In another experiment (Benson, Smith and Semenov[3]) dealing with arteriosclerosis, we fed rabbits cholesterol at regular intervals and injected bacteria into them. In still another experiment 20 years ago, before the importance of bacterial hypersensitization was fully appreciated, we injected monkeys over a period of months and found histologic changes that now can be attributed to hypersensitivity and collagen disease. We found definite round cell infiltrations and hyaline changes in the collagen tissues of the heart, blood vessels and arteries of monkeys that had been innoculated repeatedly with a streptococcus from a case of chronic maxillary sinusitis. Similar lesions have been produced by a number of workers with foreign proteins, and they attribute the histopathology to hypersensitization.

According to Rich,[33,34] bacterial allergy can be developed while the body is acquiring an active immunity to infections; thus, certain sites of predilection may become hypersensitive to the proteins of infecting micro-organisms, while others remain unaffected. When this hypersensitiveness has appeared, the affected tissue can be damaged and killed by a very small amount of the bacterial protein, one which ordinarily would be absolutely harmless.

In the *arthus phenomenon* local hypersensitivity is produced by the injection of bacterial protein. At a later time, injection of the same antigen into the same site creates a severe allergic reaction, e.g., localized edema, eosinophilic infiltration, lymphocytes and plasma cells, and hyperplasia of the connective tissue. In the *Shwartzman phenomenon* a soluble toxin may be injected elsewhere but the hypersensitivity is brief and disappears within 72 hours.

Virus vs. Bacteria. According to Feingold,[7] there are differences in the reactions that follow virus infections and bacterial infections. During the height of acute or chronic bacterial infection any existing allergy becomes worse. But during the height of a virus

infection the opposite thing happens, the allergy seemingly vanishes, only to return worse than ever when the virus infection subsides.

Comments on Combined Therapy. When an acute infection becomes chronic, some degree of sensitization to the bacteria may ensue. Once the infection is detected, the obvious thing to do is to treat it, because it may aggravate any allergic disease that the patient may have. In such a case, who has more tools to do the job—allergy and all—than a skilful rhinologist?

Large obstructing polyps must be removed. Small ones sometimes subside spontaneously. Sinuses with large amounts of viscid mucus need help, occasionally by irrigation, more often by nasal douches and capillary suction, and, at times, by intranasal window. Radical surgery seldom is necessary, even when large cysts and polyps occur in the sinuses. The methods of therapy recommended by Van Alyea[41] are up to date.

Deviations of the nasal septum, cystic turbinates and other obstructions should be treated, but with great respect for nasal physiology. As long as the offending substance cannot be avoided, specific desensitization is in order, and in addition to the common inhalants such as pollens and house dust, the use of stock and autogenous bacterial vaccines should not be overlooked.

We must treat the etiology, whatever it may be, and in hay fever the cause of the disease cannot be removed surgically. Suppurative sinusitis is different. Allergy or no allergy, psychosomatics or no psychosomatics, pus must be drained. Expert allergists and expert psychoanalysts are well aware of this fact. The recent hue and cry about the psychosomatic nose, like the mix-up concerning the psychogenic overlay in deafness, which nearly always has an organic basis that can be measured with some degree of accuracy (Semenov[37]), reminds me of an old riddle. Which came first—the somatic egg or the psycho hen?

Without going any further into the pitfalls of overlapping diagnoses, it may perhaps be well to return to the subject of nonsuppurative inflammation because so many cases of allergy fit into this category.

In the early stages (stage I), chronic nonsuppurative inflammation of the nose and sinuses may simply be called catarrhal inflammation. Catarrhal inflammation, of course, is seen in acute rhinitis, and also during the stage of recovery (final stage) of an empyema of the sinuses, so it is not always allergic in origin.

STAGE I

Catarrhal Sinusitis

The sinus mucous membranes, as their name indicates, are moistened by a mucus-containing secretion, which is derived mainly from the epithelium. Under normal conditions a small inconspicuous quantity of mucus is formed, but with the advent of catarrhal inflammation, a heavy mucoid discharge appears, and, on account of its great viscosity, it moves slowly, may stagnate, and causes a number of unpleasant symptoms. Inasmuch as there are very few glands in the sinuses, most of the mucus obviously is formed by the surface epithelium (fig. 31). Histologically, there is an infiltration of lymphocytes, and in allergy, eosinophils but no pus or fibrin is found. The mucosal thickening is minimal (1 mm. or less), in keeping with the hyperemia and edema.

In mild cases the pathology may be latent and asymptomatic. On the other hand, there is no denying the existence of a great number of cases in which the etiology is obscure. The exudate is expelled with difficulty and sometimes accumulates in the sinuses.

Mucocele of the Paranasal Sinuses

A mucocele is a large accumulation of pathologic mucus in a closed cavity, such as a frontal, ethmoidal, sphenoidal

or maxillary sinus. The main cause is obstruction of the outlet. In the frontal sinus, which is the one most frequently involved, we have very few glands; therefore, the mucus stems from the surface epithelium, and lacking an outlet, it fills the sinus to such an extent that there is retention and eventually pressure atrophy. The thin bony wall (orbital plate) finally softens, sometimes with great external deformity and intracranial involvement. Secondary infection of the mucocele is known as "pyocele," a more serious condition owing to the presence of pus and bacteria.

The frontal duct or sinus outlets may be obstructed in a number of ways: (1) inflammation and swelling, (2) scar tissue resulting from surgery or injury, (3) osteoma, (4) polyps, or (5) deflected nasal septum. In short, any condition that will block the ostium for a long period of time will prevent the escape of the mucus. After radical surgery of the frontal sinuses, there is the possibility of developing a mucocele from remnants of epithelium that are permitted to remain in the cavity at some distance from the outlet. Such epithelial remnants frequently produce cysts because they become buried in scar tissue. The symptoms and signs of mucocele are largely those of pressure and deformity of the surrounding structures. Roentgenograms may reveal (1) enlargement of the sinus cavity, (2) obliteration of the scalloped or septate outlines, and (3) rarefaction and pressure necrosis.

FIG. 31.—Catarrhal sinusitis. The term "catarrh" is a good one for this condition. Note the folding of the epithelial surface, a common condition when much catarrhal inflammation is present. It would almost seem that the surface epithelium tries to outdo itself to produce mucus. (Semenov[23])

STAGE II

Hyperplasia

Hyperplasia, an increase in the number of cellular elements, always produces a tissue that is similar to the original part; accordingly, hyperplasia of the epithelium gives rise to new epithelial cells, and that of the connective

tissue to new connective tissue cells. In long-standing cases of latent sinusitis, in which there is good reason to believe that some hypersensitivity exists, one finds on histologic examination that the connective tissue layers contain an increased number of fibroblasts. Insufficient emphasis has been accorded to the collagen changes; as a consequence, their importance may have been underestimated and possibly completely misinterpreted.

Allergic sinusitis, a form of hyperplastic sinusitis that is the result of allergy rather than the cause of it, occurs in about 20 per cent of hay fever and/or asthma cases, and often is asymptomatic. Histologically, typical cases show mucinous epithelium, collagenous thickening, hyalinization of the basement membrane, eosinophils, lymphocytes and plasma cells. In *atypical cases,* our most important clue, the eosinophilia, may be insignificant or due to something else. Sooner or later infection is added to existing allergy, and the resulting inflammation creates a new situation which often is severe and stubborn.

The pathology sometimes defies etiologic classification, as the following analysis of 500 cases indicates:

	PERCENTAGE
Allergic	17
Non-allergic	47.6
Equivocal or uncertain	35.4

Allergic sinusitis often is associated with radiographic signs of slight rarefaction of the bony walls of the sinuses, especially the thin ethmoidal partitions. Inasmuch as the polypoid changes are characterized by a tremendous amount of chronic edema, it is possible that some degree of pressure atrophy may explain the bony changes.

The extreme degree of swelling may be called polypoid sinusitis, while a lesser degree is generally called hyperplastic sinusitis. Although allergy is a common cause of both conditions, it is not the only cause. Infection may lead to similar changes. In either event, the salient feature is the edema. In chronic cases, edema and hyperplasia are so closely allied that one is seldom seen without the other, and various terms (chronic hydrops mucosae, polypoid thickening and polypoid degeneration) have been used to describe the gross appearance of the tissues.

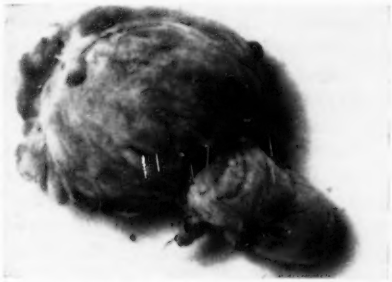

FIG. 32—Large polyp in the antrum. The lining has been everted and packed with cotton which is not visible in the picture.

Histologically, the edema and increase in collagen substance, the hyperplasia of epithelium and of connective tissue, and infiltration of leukocytes, eosinophils, plasma cells, lymphocytes and round cells may be seen in various proportions. The polypoid appearance is due to the combined effects of edema and connective tissue hyperplasia. The actual hyperplasia becomes evident when the edema subsides. As stated, under normal conditions there are very few goblet cells in the epithelium and no signs of edema or round cell infiltration in the submucosa. The epithelium varies from 25 to 50 micra in thickness, but usually it is about 35 micra in height; when it exceeds 50 micra, it may be considered hyperplastic. The basement membrane is extremely thin. Hyalinization is a sign of pre-existing inflammation, usually allergic. Hyalin

thickening of the basement membrane is characterized by the appearance of a pink homogeneous layer under the epithelium, and when it becomes 10.0 micra in thickness it is pathological.

The increase in thickness of the membrane, therefore, is caused partly by an increase in the collagen elements, partly by the edema, and partly by the proliferation of new cells. Even though we arbitrarily divide the pathology into exudative, proliferative and degenerative stages, the main feature is the amount of permanent thickening.

The exudative changes may be circumscribed or diffuse, involving either localized areas or the entire lining. They may come and go, creating mucus or mucopus, and discharge from time to time, but they are reversible. The hyperplasia is not, and in the few cases in in which I have used it, ACTH and cortisone simply reduce the exudation and edema but do nothing for the hyperplasia and degeneration.

In stage two, therefore, we have a proliferation of epithelial and mesenchymal elements and lymphoid tissue elements, an increase in the number of spindle or stellate-shaped fibroblasts, and an increased amount of collagen, which in the nose and sinuses tends to form polyps, polypoid or myxoid tissue.

Hyperplastic sinusitis often is latent and may exist for a great many years without giving rise to any symptoms. It is diagnosed by roentgenography and by washings from the cavity, which usually reveal catarrhal exudate upon cytologic examination. In asymptomatic cases, treat the allergy and do not touch the sinuses.

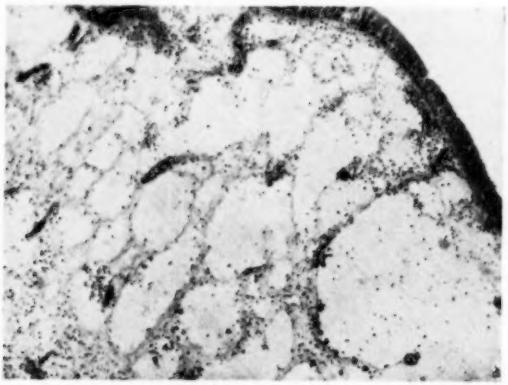

FIG. 33—Allergic sinusitis showing the reaction of the connective tissue, the swelling and scattered inflammatory exudate. (Semenov[24])

The mucous membrane becomes 20 times its normal thickness (fig. 6). It has a pale yellowish or greyish color, and the surface is covered with more or less uniform swellings (wheal-like distentions). Sessile polyps begin in this manner, and when the tissues prolapse, an actual stalk forms and the polypus hangs into the nose (fig. 44). The tissue spaces may rupture and form cystic cavities filled with tissue fluid or lymph. At the same time the glands that are present begin to dilate and become cystic (fig. 21). Thus two types of cysts arise: (1) serous cysts and (2) retention cysts. The serous cysts contain a watery fluid and the retention cysts contain thickened mucus. Either one may become infected.

As stated in previous publications,[36] nearly half of the cases of hyperplastic sinusitis are allergic. The connective tissue in a sensitized state is capable of reacting in a peculiarly intense way when it is brought into contact with any antigen toward which it has developed an allergy. The antigens may be bacterial or nonbacterial, and in either event there is intense edema, degeneration of cells and intercellular substances on the one hand, and proliferation of new cells and intercellular substances on the other (fig. 33).

Lymphoid follicles. In 1929 and in 1934, we mentioned follicles of lymphatic tissue in hyperplastic sinus mucous membranes, and suggested at the time that these structures might be products of the connective tissue itself, i.e., polyblasts or undifferentiated mesenchymal cells.[35]

Lymphatic nodules are never seen in healthy sinus mucous membranes, and when present, either in the nose or sinuses, must be due to a chronic infection or allergy, or possibly, and this seems more likely, both conditions. They always are confined to the tunica propria, where they form typical secondary follicles, i.e., a light center composed of large pale cells (the size of immature lymphocytes) surrounded by a very definite ring of darker staining mature lymphocytes (fig. 34).

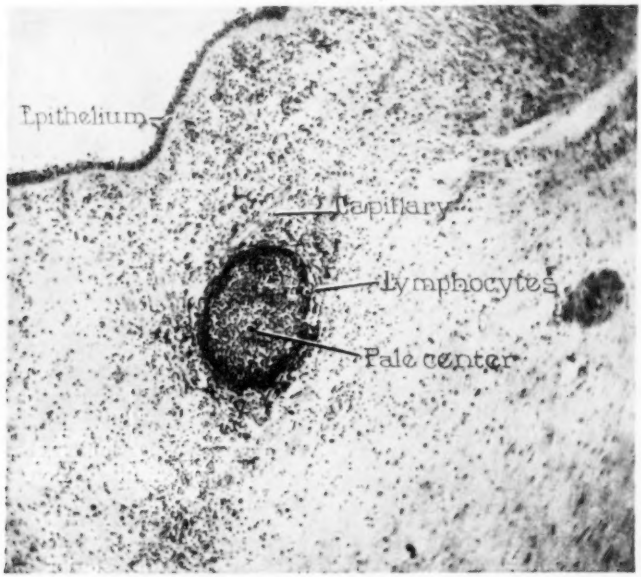

FIG. 34—Lymphoid follicle in chronic hyperplastic sinusitis. These follicles are exactly like the ones seen in tonsils and are composed of a ring of lymphocytes around a pale center composed of large lymphocytes and reticulo-endothelial cells. The center is called the secondary nodule. (Semenov[34])

The meaning of these new-formed follicles is not clear, but I think that they probably are inflammatory reactions of a special sort, because I found them twice as often in the cases that were definitely allergic. We often find many hyperplastic follicles in the throats of allergic children, and of adults as well. Although I like the term "follicular

pharyngitis," I cannot say the same for the term "follicular sinusitis," even though others have used it since I first reported the histologic formation of lymphatic follicles in the tissues of the nose and sinuses. They cannot be diagnosed clinically. I find the follicles in only about 3 per cent of my material, which embraces well over a thousand specimens, and in any given case only a solitary follicle may be encountered. What connection, if any, do they have to the growth and involution of lymphoid tissue as it relates to the adrenal glands (White[44])?

Chronic Rhinitis

Chronic catarrhal inflammation and chronic hyperplastic rhinitis, two more or less inseparable conditions, one being a more advanced form of the other, differ mainly in the amount of mucosal hyperplasia and thickening. In typical cases of hyperplastic rhinitis, we encounter, in addition to catarrh, infiltration of round cells and plasma cells, which may be focal or diffuse, a great deal of hyperplastic thickening and chronic edema, which always centers in the subepithelial layer of stroma. The blood vessels and the cavernous plexuses of the deeper layers become dilated and their smooth muscle fibers show some evidence of alteration, owing presumably to the chronic bouts of congestion. The epithelium becomes stratified. The basement membrane often is thickened and hyalinized. The glands are surrounded by periglandular cellular infiltrations and seem to become larger. Their ducts often are dilated and cystic. These changes frequently affect the posterior ends of the turbinates, sometimes the entire middle turbinate and ethmoidal region, and in severe cases the entire nose. As has been stated, the principal lesion is the edema in the upper layers of the areolar connective tissue. Spraying with vasoconstrictor drugs produces only a slight degree of shrinking. The swelling forms a pale redundant area of thickening in the above-mentioned areas.

Thus, the term hyperplastic rhinitis refers to the permanent increase in thickness rather than to the edema which may come and go. Both conditions are so closely related, however, that one is seldom seen without the other, especially in perennial allergic rhinitis.

Hypertrophic rhinitis is a milder form of chronic rhinitis. Vasoconstrictor sprays cause the congestion to disappear, which rules out true hyperplasia.

Polypoid rhinitis is a more advanced form of hyperplastic rhinitis. The intense edema which characterizes this condition accounts for the development of gross thickening in some areas, and in other areas actual polyp formation. Polypoid changes are commonly encountered in the ethmoidal region and sometimes result in degeneration of the middle turbinate. The pathology is easily recognized in this location, since a large part of the ethmoidal area is visible on direct rhinoscopy. Similar changes within the sinuses can be demonstrated only by roentgenography.

Polypoid turbinate tips. Polypoid swelling of the posterior ends of the turbinates is an exceedingly common condition. Indeed, it is the most frequently encountered manifestation of chronic rhinitis that we have, and it may be attributed to a number of different causes. It is seen with and without sinus infection, also with and without nasal allergy, the natural inference being that numerous anatomic factors probably enter into the etiology, e.g., the bottleneck at the posterior choanae. A rather obvious factor is the constant flow of acrid mucus that passes the bottleneck, not to mention the heavy traffic that comes with acute bouts of nasopharyngitis. Chronic nasal allergy, of

course, is a very important factor as demonstrated in the numerous specimens that I have examined, but it does not explain all of the cases.

The histopathology is exactly like that of hyperplastic rhinitis in general, except for the following feature: the boggy distended bulbous masses sometimes are called "mulberry tips" owing to their papillary appearance (fig. 35). This increase in surface area occurs in all catarrhal membranes but is more evident here.

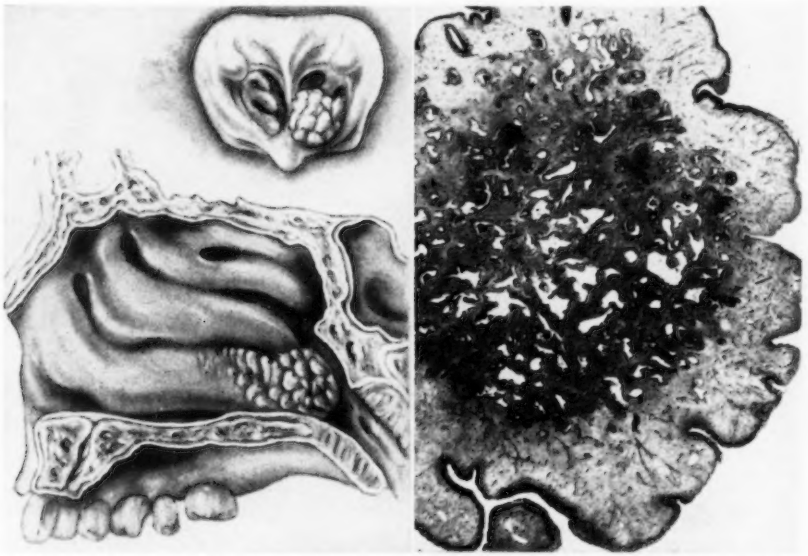

FIG. 35—Posterior tip of inferior turbinate showing polypoid swelling, a common condition which in extreme cases creates the so-called "mulberry tip." The histopathology is similar to that of polypoid sinusitis except that the superficial layer of stroma is involved in the nose. Note the sharp line of demarcation between the swelling of the upper layers (pale area) and the central core of the turbinal mucosa. The core contains many cavernous blood vessels. This erectile tissue sometimes is mistaken for angioid tissue and diagnostic errors are not uncommon when general pathologists fail to understand the histology of the nose. (AFIP 86277, X 10)

Histologically, mucoid degeneration of the epithelium, hyalinized basement membrane, edema of the upper layer of stroma, many blood vessels (cavernous tissue) and glands in the deeper layers, and papillary formations are characteristic of polypoid turbinal tips. Specimens removed surgically and sent to pathologists for diagnosis should be properly labeled, indicating the source of the tissue, otherwise an erroneous histologic diagnosis of "angioid polyp" is apt to be made because of the great vascularity of the turbinal tissues.

Chronic allergic rhinitis is a frequent condition and accounts for two-thirds of the chronic perennial rhinologic complaints. The atypical forms sometimes cannot be separated from the catarrhal or hyperplastic changes that belong to nonallergic rhinitis. In typical cases, the turbinates and ethmoidal area present pale, wet, boggy swellings of the mucosa. The enormous "water-logging" of the fibrillar stroma is striking in histologic sections. The basement membrane is thickened and hyalinized, and the changes in the tunica propria are most marked in the upper (subepithelial) lay-

ers, while the deeper layers are less swollen (fig. 36). The eosinophilic infiltration is distinctive and always associated with lymphocytes, plasma cells and an increase in the number of fibroblasts. When the tunica propria can no longer restrain the edema, it prolapses and becomes polypoid. This usually happens in the ethmoidal area where the mucosa is thin and weak. Where the edema is an important factor in all three conditions. ACTH and cortisone did not reverse the degenerative pathology in the case that I have treated.

Cystic changes were found in one-third of the specimens of chronic sinusitis[36] and appeared in half of the cases in which the mucous membranes were more than 2.0 mm. in thickness. Cystic dilatation was seen in 85 per cent of the

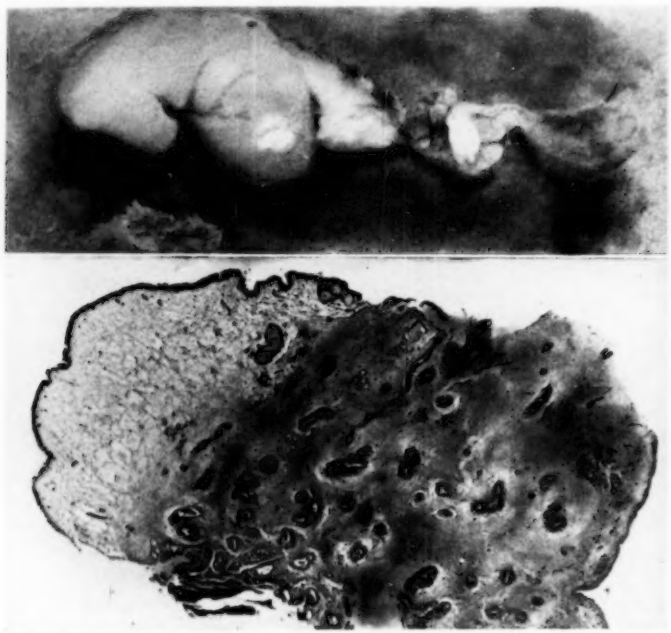

FIG. 36.—Polypoid degeneration of the middle turbinate—allergic. The edema is largely confined to the subepithelial stroma. Note the cavernous vessels in the deeper stroma, which is more fibrous. (10 X).

connective tissue is firm, as on the septum and the inferior turbinate, it simply forms a mass of polypoid tissue but no true polyps.

STAGE III

Cysts and Polyps

The degenerative stages include three more or less irreversible lesions: (1) retention cysts, (2) serous cysts, and (3) pedunculated polyps. Chronic allergic cases. Deep seated microabscesses, due to infection of the small cysts, were found in 10 per cent of the cases of purulent chronic sinusitis.

1. *Retention Cysts.* Obstruction of the gland ducts by leukocytic infiltration or by edema initiates the formation of retention cysts, and as each acinus dilates it encroaches on the adjacent ones and eventually the dilated tubules

coalesce to form a large cyst (fig. 19). The interior of a large retention cyst is lined with epithelium in various stages of atrophy, and areas of poorly preserved or flattened cells gradually pass over into well preserved glandular cells, the epithelial gradations being stages in the cystic evolution. Retention cysts contain a thick mucoid material which sometimes is mixed with concentric deposits of epithelial debris and leukocytes.

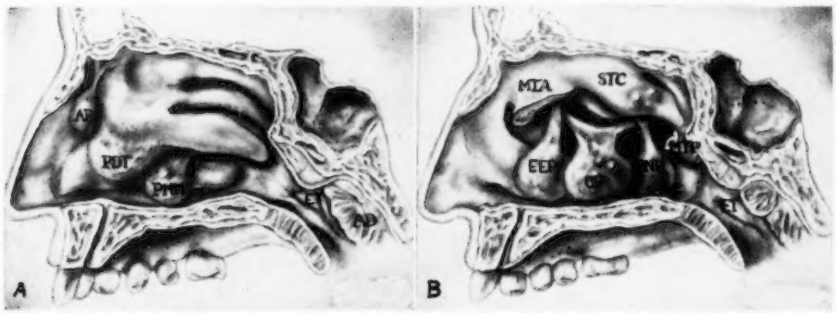

FIG. 37—(A) Lateral wall of the nose showing origin of nasal polyps. AP—anterior polyp arising from the ethmoidal area; PDT—polypoid middle turbinate (broad base sessile polyp); PMM—polyp in the middle meatus. (B) Lateral wall of the nose with middle turbinate removed to show origin of polyps. MTA—middle turbinate anterior attachment; EEP—edematous ethmoidal polyp; CP—cystic polyp springing from a gland bearing area; PNP—postnasal polyp arising farther back; STC—superior turbinate cystic polypoid swelling; MTP—middle turbinate posterior attachment; ET—eustachian tube (edematous).

2. *Serous Cysts.* When the tissue spaces of the loose areolar stroma become extremely edematous, they frequently form large cystic cavities. These cysts have been called nonsecreting cysts, interstitial cysts and mesothelial cysts and are not lined with epithelial cells. They are thin-walled and in the absence of suppuration are filled with a clear amber fluid that will clot after it is washed out of the antrum and stands for 15 or 20 minutes. As a rule, only one antrum is involved, and I do not think allergy plays an important part in these cysts.

Serous cysts can become very large and often obstruct the sinus outlet. They cause much pain and I treat them through an antrum window opening—simply rupture the bag. We used to resort to Caldwell-Luc exenteration, and that is how we obtained the rare photographs shown here, but that was twenty years ago—before we understood the surgical pathology.

NASAL POLYPS

In the minds of the majority of general practitioners, and even some pathologists, the cause of nasal polyps is an enigma. The descriptions that one finds in some textbooks are not as accurate as they might be, and one still reads misconceptions that have been copied from one book to another. It is little wonder that the average medical student ends up with a rather hazy notion of the subject. The confusion is heightened by the use of obsolete terms, such as myxoma, fibroma of the nares, fibroma edematodes simplex, adenofibroma edematodes, fibroma edematodes cysticus, etc. Some of this time-honored terminology dates back to clashing opinions regarding the classification of nasal polyps. Those supporting the tumor theory based their arguments on the continued recurrences. The other school, denying malignancy, based its views on the fact that polyps never form metastases. Although the latter theory proved to be correct, we must always remember, in dealing with polyps, to look for signs of malignancy, because more than one unsuspected cancer has been found lurking behind them.

The subject of nasal polyps is as old as the history of medicine itself, and nearly every authority, from Galen on down, had

something to say about this subject. The list of names mentioned by Jonathan Wright[46] could very well pass for a "Who's Who in Medical History." A pathologic condition that has been recognized and treated for so many centuries certainly merits more than passing mention at this time.

The derivation of the term itself is lost in antiquity, although its Latin and Greek roots mean "many feet," which serves to indicate that the ancients were well aware that they are seen in the nose more often than in the sinus. He described the polypoid lower borders of hyperplastic inferior turbinates. Alexander Monro also made some remarkable observations nearly two centuries ago and showed an insight far ahead of the time in which he lived.

Within recent years we have learned that nasal polyps are frequently due to

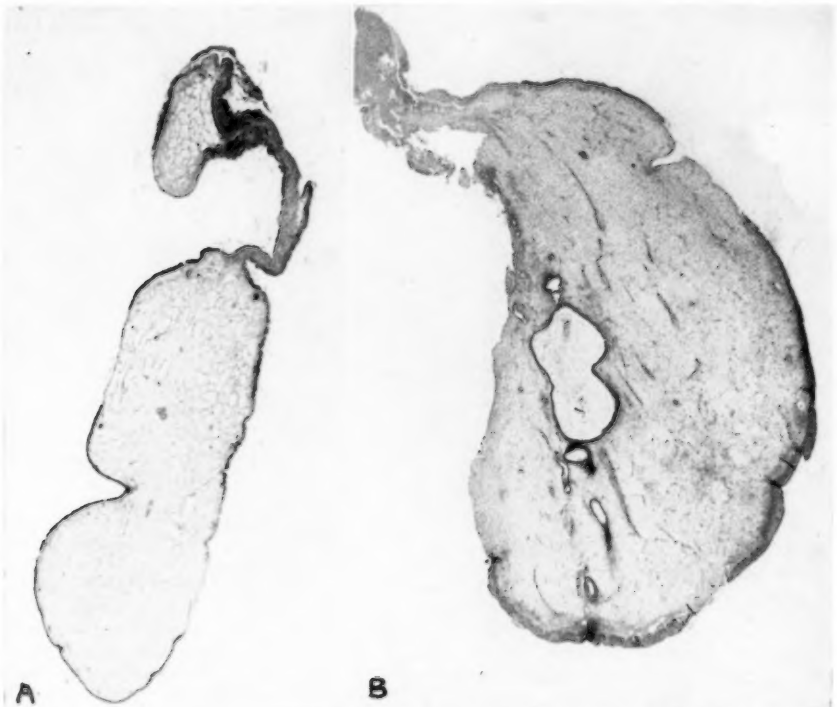

FIG. 38.—(A) Simple edematous nasal polyp. Note the reticular appearance of the distended stroma. This polyp came from an area that was devoid of glands. (B) Nasal polyp containing cystic glands. This one is more myxoid. Both are pedunculated. (10 X)

of the fact that when one removes a polyp, he is apt to find many others behind it and that recurrences are common. As time went on, almost anything that hung down in the nose was called a polyp, even a piece of wood covered with granulations. Monstrous errors of this type filled the literature for hundreds of years, but here and there we find fairly accurate observations. Morgagni, for example, in speaking of polypi said 200 years ago allergy or an inflammation that causes prolonged edema of the mucous membranes. The etiology, therefore, includes infection and/or allergy. It has been known for some time that polyps favor certain areas of the nose, e.g., the middle and superior meatuses, and that the mucous membrane in these areas is

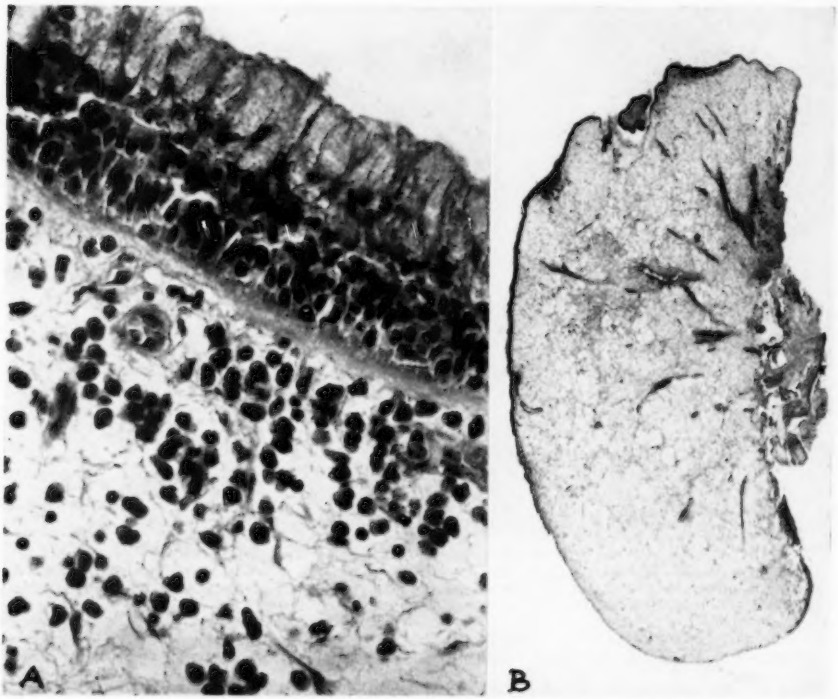

FIG. 39—The patient, age 22 years, had perennial allergic rhinitis and asthma with polyps hanging down to the nostrils. There was an extreme degree of deflection of the nasal septum. He was sensitive to grasses, ragweed, feathers, horse dander, and gave a four plus reaction to house dust. He was treated with desensitization, polypectomy and submucous resection of the septum. In (A) the hyperplastic epithelium is of the catarrhal type and stratified. Note the thickened basement membrane (600 X). (B) Polyp from the middle meatus (10 X). Note the edematous stroma and spread of the blood vessels. No glands are seen in this specimen. The sinuses were normal.

different in structure. Zuckerkandl[47] was the first to emphasize the differences in the ethmoidal capsule and mentioned the fact that the mucosa was exceptionally thin, and that the underlying stroma was exceedingly delicate and loose. The mucous membrane in these situations reacts to inflammation and allergy by becoming exceedingly edematous. When this happens, it is apt to prolapse and form a polyp, which persists as long as the inflammatory condition continues. At one time there was a school of thought which held that the pathology was due to an osteitis or periostitis of the underlying bone. As recently as 1925 Freedman[11] advanced this notion. As a matter of fact, the osseous changes are secondary to the polypoid edema, not the cause of it.

A typical nasal polyp, "mucous polyp" or "edematous polyp" frequently is shaped like a lima bean and has a grayish or yellowish color. It always is smooth, soft and shiny, and has a translucent wet pulp much like that of a grape. It is not a true tumor, but merely a swollen mass of thickened connective tissue. In short, it may be due to the edema of a chronic sinus infection and/or perennial nasal allergy.

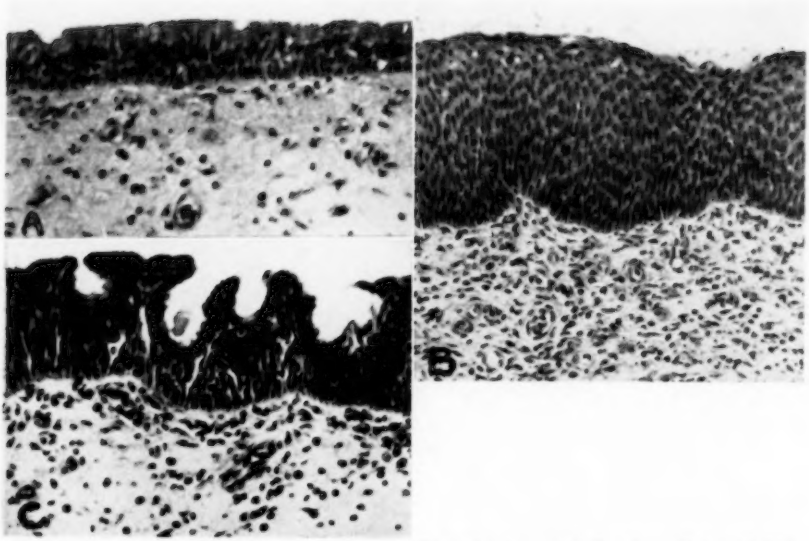

FIG. 40.—Variations in the appearance of the epithelium of polyps. (A) Pseudostratified epithelium. (B) Hyperplastic multilayered epithelium on another side of the same polyp as (A), which was infected. Note absence of basement membrane in this specimen. (C) Hyperplastic epithelium assuming weird forms. (205 X).

When the edema subsides, the polyp may disappear, especially when a broad base (sessile polyp) is present. On the other hand, a pedunculated polyp with a narrow stalk is inclined to persist and grow to a considerable size.

For all practical purposes, nasal polyps are caused by either infection or allergy. Further subdivision into adenomatous types, cystic types, etc., may be of some interest to pathologists and museums but have little meaning elsewhere. When the polyps happen to spring from a portion of mucosa that normally is rich in glands, the glandular elements appear in the polyp (fig. 38). The glandular elements may or may not increase in number, and sometimes there is infolding of the epithelium.

Cystic polyps spring from gland-bearing areas, but in this case the gland outlet becomes obstructed and forms retention cysts. At times the infolded surface epithelium forms crypts that sometimes become blocked.

In children one frequently sees polypoid swelling of the middle turbinate, but many years may elapse before pedunculated polyps appear. This raises an interesting question. Why is one condition so common and the other so rare in children? Does the connective tissue lose its elasticity with age?

Nasal polyps arise in the nose itself and nearly always spring from the lateral wall of the nose—particularly the upper half (fig. 37). Some are attached to the middle turbinate, others to the margins of the hiatus semilunaris, under cover of the concha. The latter tend to appear in and around the middle meatus. Polyps can occur in other parts of the nose, however, and in an extreme case the entire nasal mucosa, including the nasal septum, may become polypoid.

As a rule, the epithelium is heteroplastic, although normal respiratory

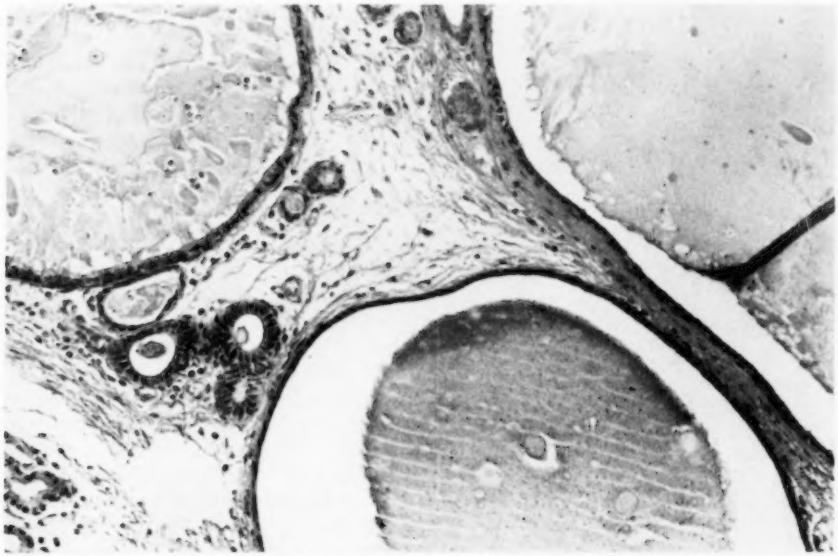

FIG. 41—Cystic nasal polyp (230 X). Note the mucus in the cysts and flattening of the epithelium. The patient, age 25 years, had unilateral nasal obstruction and discharge for three months. He denied symptoms of hay fever and asthma. Roentgenograms of the sinuses were normal. Intradermal tests were negative except for a questionable reaction to house dust. The pathologic condition was unilateral and confined to the nose, and the polyp was pink in color. It was studded with cysts which exuded mucus at the time of surgery. The polyp was a long one (2.5 inches) and attached in the spheno-ethmoidal area. Histologically, the epithelium was slightly hyperplastic, the basement membrane thin and the stroma fairly firm and fibrous. The cause of this polyp is open to speculation. It may be a true adenomatous type of lesion. The absence of sinus infection and questionable allergy history and tests makes this case a problem for the experts. Not all polyps are allergic or due to infection.

epithelium may be found over large areas. In some the cilia are lost through mucoid degeneration or trauma; and in others the epithelium develops to such an extent that it is stratified (fig. 39), and even metaplastic. This may be due to many different causes, the effect of air currents, mechanical irritation, catarrhal inflammation, etc. The basement membrane often is thickened, especially in the presence of allergy.

The connective tissue is gelatinous and waterlogged in character, and in histologic sections one may see many young spindle and stellate connective tissue cells, which give it an immature "myxomatous" appearance.

There are three easily recognized changes: first, a prominent cellular and intercellular edema; second, a proliferation and hyperplasia of fibroblasts; and, third, hydration and degeneration of the collagen fibers. The injury and reaction of connective tissue cause some interesting effects. Even in adults, one type of connective tissue may be directly transformed into another. As a consequence, cartilage, lymphatic follicles and other mesenchymal tissues sometimes develop in nasal polyps.

Polyps are not very vascular; that is, the blood vessels that normally occur in the region from which they spring simply stretch out. The capillaries in the loose waterlogged stroma are pulled far apart by the distention and ballooning of the tissues.

The leukocytic exudate is scattered throughout the tissues, but here and there eosinophils, round cells, and

FIG. 42.—Cystic nasal polyp (AFIP 117141, X 5), low-power view of case discussed in figure 41.

plasma cells tend to congregate (1) just under the epithelium, (2) around the glands, and (3) around the blood vessels as perivascular collars.

Inasmuch as polyps may be due to either allergy or infection, and sometimes even cancer, they should be classified according to etiology rather than morphology. Our attention always must be directed to the cause of the polyps, because this will influence our treatment (figs. 41 and 42).

Antronasal polyps arise from the mucosa of the maxillary antrum. They extrude through the accessory ostium or sometimes through the natural ostium to enter the middle meatus and drop back along the floor of the nose (fig. 43). As a rule, this type of polyp is pear-shaped and does not have the mulberry appearance of an enlarged inferior turbinate. I have seen ten antronasal polyps and all of them were due to sinus infection. All were unilateral and, in my experience, did not return after they were removed. In most instances amputation of the polyp and an antrum window operation were sufficient. Allergic polyps, on the other hand, are bilateral and arise in the nose itself. They often return at frequent intervals. The chief difference between the two kinds of polyps, therefore, is in the treatment and more favorable prognosis offered by the antronasal or antrochoanal type. This does not mean that all polyps that begin in the antrum and extrude through the ostium to hang down into the nasopharynx are due to infection. Some are associated with allergy, but not as many as the current literature would lead one to think. The origin of the particular polyps that I am attempting to define frequently is on the lateral wall of the antrum (fig. 44), and I have often wondered why this should be so. One would ordinarily expect them to spring from a point somewhere nearer the ostium where the greatest amount of irritation and discharge occurs. The answer, I believe, may lie in the histology and embryology of the antrum. The lateral portion (neosinus) is extremely thin and delicate, and it is subject to much edema because of the long line of communication of the lymphatic drainage. As a rule, polyps that arise in the neosinus rarely, if ever, have glands in them (fig. 45).

As stated, they are unilateral, and the narrow pedicle may arise from the posterior, superior or lateral wall of the antrum. Histologically, the polyp is a simple one, consisting mainly of dis-

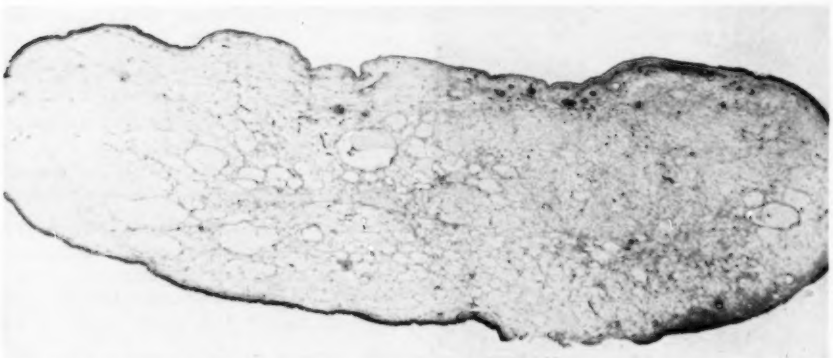

FIG. 43—Antronasal choanal polyp (10 X). This 29-year-old patient complained of unilateral nasal obstruction for three months. The polyp hung down to the level of the soft palate. Roentgenograms revealed a unilateral maxillary sinusitis on the same side. At the time of surgery the stalk was traced to the ostium of the antrum. The polyp was removed with a snare and an antrum window made under the lower turbinate. Two smaller polyps extruded through the opening and were removed with punch and snare. The patient made a rapid and uneventful recovery. Histologically, the cross section of the polyp is oval, very edematous and devoid of glands. Pus was present in the nose and infiltration of tissues consists mainly of lymphocytes, plasma cells, a few neutrophils and only a scattering of eosinophils. The naso-antral wall was thin and soft, and the cavity filled with mucopus. This case was classified as a sinus infection rather than allergy. Allergy tests were negative, the scant eosinophilia means nothing, but the response to sinus surgery was excellent. Diagnosis is based on the entire symptom complex, not one sign alone.

tended edematous stroma with a scattering of round cells. In typical cases there are no eosinophils. This does not mean that allergy does not occur here, but rather that it is coincidental and rare in unilateral, solitary polyps. The cause of such polyps may be mechanical —e.g., aerosinusitis.

According to Heck, Hallberg and Williams,[13] approximately 3.7 per cent of the nasal polyps at the Mayo Clinic were of the antrochoanal type. They found eosinophils and other signs of allergy in about 20 per cent, and, after reviewing their excellent data, I am inclined to believe that our observations

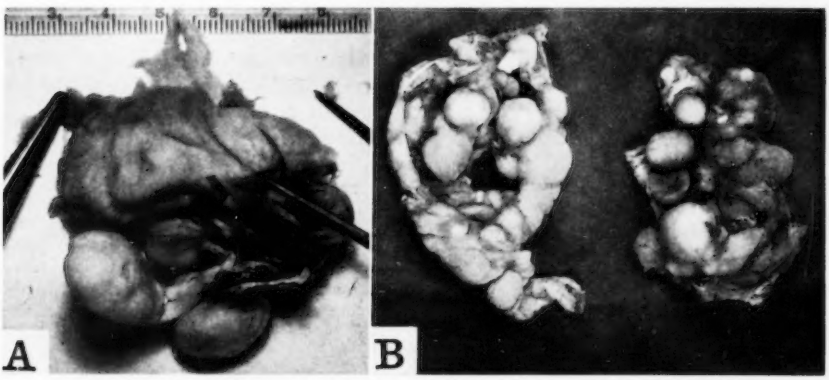

FIG. 44—(A) Antronasal choanal polyp and entire maxillary lining removed by Caldwell-Luc operation. The polyp with the compressed stalk was hanging in the nose, the other one was confined to the antrum. (B) Multiple infected cysts in a bilateral maxillary sinusitis. The cysts were filled with mucopus. In cases such as these the decision for or against radical surgery is a hard one. (Semenov[36])

FIG. 45.—Diagramatic representation of the origin of antronasal polyp shown in figure 44 (A). The lining of the antrum is thickened and polypoid. The polyp protrudes through the sinus ostium and hangs down into the posterior part of the nose and choana.

are similar even though interpretations may differ.

Bilateral Nasal Polyposis

Atopy usually leads to bilateral polyposis and, as a rule, a bilateral pansinusitis, because the underlying cause is constitutional in nature. The lesions may be more pronounced in one anatomic area than in another, and, in addition to histologic factors, such as differences in the development of the tunica propria, we must remember the impact of inhalants is greater in the middle and superior meatuses. As Proetz[32] has shown, the inspired air arches upward and strikes this area first. It is reasonable to suppose that pollens and other inhalant allergens bombard this area more than they do other parts of the nose. Needless to add, once the polyps form, the air current is deflected in another direction. Regarding the direction of the air current in the nose, the exhaled stream passes along the inferior meatus, according to Proetz, and I have seen a small anterior inferior perforation of the septum whistle only on the outgoing tide, i.e., expiration.

While at the Armed Forces Institute of Pathology I examined 160 polyps, and two-thirds of the specimens seemed to be allergic in origin. In the remaining third, the pathology was considered nonallergic. One out of every four polyps contained cystic glands. Three polyps were of the choanal type. Lymphoid follicles were seen in only four specimens in this series. Walsh and Lindsay[42] also concluded that some polyps were associated with allergy and others with infection, that the latter are unilateral and that their removal is followed by better results than in the allergic cases, which are frequently bilateral.

APPLIED PATHOLOGY AND THE FUTURE

Nearly all of the pathology cited so far is from cases treated before the era of chemotherapy. Can this information guide us forward to a newer and better understanding of therapy?

The term "mucous polyp" has no special meaning. As stated, the pathologic lesions center in the connective tissue, and the morbid changes might be classified with the collagen diseases. One layer, the tunica propria, seems to suffer the most injury, owing presumably to the character of its mesenchymal cells and intercellular substances. Whether or not hyaluronic acid, a constituent of the cement substance which functions as a barrier to the spread of fluids, may shed some light on the question is something to wonder about.

Hyaluronic acid, a mucopolysaccharide acid that is found in almost all tissues, functions as a tissue cement, holding cells in a jellylike matrix and also bonding tissue fluid. The gels that it forms seem to play a part in the exchange of fluids and metabolites. The viscosity of this gel determines its effectiveness as a barrier.

Hyaluronidase is one of the enzymes. It is found in many different tissues, in snake and bee venom, some strains of streptococci and pneumococci. It lowers the viscosity of hyalu-

ronic acid and is known as "the spreading factor." What, if any, part do these biochemicals play in polyps, which, after all, look like gelatinous masses to the naked eye?

Reports dealing with general pathology suggest a defect in the process of polymerization of hyaluronic acid. It is said that the effect of hyaluronidase is increased by desoxycorticosterone and abolished by cortisone; that cortisone apparently causes a delay in the development of all of the connective tissue elements at an elementary level; that it may block connective tissue hypersensitivity; that desoxycorticosterone has a stimulating effect on fibroblasts, but cortisone inhibits fibroblast proliferation; and that certain persons secrete an excess of one hormone in relation to the other (Selye[40]). With these thoughts in mind I have attempted to see what happens when cortisone is injected directly into polypoid tissue, and the following case report, one of a series of cases, is cited as an example.

CASE REPORT

Mrs. L. H., age 30 years, has a perennial allergic rhinitis and sinusitis that for 20 years has resisted desensitization therapy, nasal septum and sinus surgery, innumerable polypectomies, and, at the Mayo Clinic, roentgen therapy directed at the polyps. Everyone who has ever seen her has been impressed with the polyposis of her entire nasal cavity, including the nasal septum, which is almost an inch in thickness. She has complete and total obstruction.

Shrinking agents and antihistaminics are of little avail. Aspirin in any form causes an immediate and violent allergic swelling of the nose and face. Procaine, alcohol and anger will cause a similar reaction but to a lesser extent. Fortunately, she can take antibiotics, and when aureomycin is used, the inflammatory edema subsides somewhat, but not enough to give her any breathing space.

She has the most extensive polyposis that I have ever seen or heard of. There is a thick, brownish nasal discharge that reminds me of rubber cement. All of the sinuses are opaque and show x-ray evidence of being expanded by the pressure of the polyps. Polyps have been removed on numerous occasions, and upon histologic examination they have the typical appearance of infection and allergy combined.

Her chief complaint is headache and nasal obstruction. She cannot get any air in or out of her nose and has no way of blowing it out. The eustachian tubes are normal in appearance, and there is very little adenoid tissue in the nasopharynx, possibly due to the radiation therapy mentioned above. Her general health is good—B.P. 120/80.

Laboratory Findings:

Nasal smears:

Large number of pus cells, bacteria and eosinophils.

Bacteriologic cultures of the nose and sinuses:

Contain a mixture bacteria: Pseudomonas aeruginosa; Diplococcus pneumoniae; Staphylococcus aureus and albus; Neisseria catarrhalis; and gram-negative bacilli of the coliform group.

Thorn Test:

Eosinophils before adrenalin:	438/cm.
Eosinophils 4 hours after adrenalin:	133/cm.

Blood Count:

Erythrocytes		5,900,000
Hemoglobin		101.8% (15.8 gm/100 cc.)
Color Index		0.88
Leukocytes		7,800
Neutrophils		54%
Filamented	53%	
Nonfilamented	1%	
Eosinophils		6%
Basophils		0.5%
Lymphocytes		35%
Monocytes		4.5%

Skin Tests:

Intradermal dust (1/400) — 2 cm. wheal and 3.5 cm. flare

Autogenous bacterial vaccine—strong positive reaction (delayed type)

Diagnosis:

1. Perennial allergic rhinitis and sinusitis, chronic, twenty years duration, severe.
2. Polypoid degeneration of the entire nasal mucosa, all of the sinuses, and entire nasal septum.
3. External deformity of the nose, due to pressure of the polyps.

CORTISONE TREATMENT

Authorities agree that cortisone has the advantages of topical application, particularly in diseases that follow a chronic course such as this one.

If it is any good at all, this is the case that needs it. Topical packs, sprays and drops were tried without result. Then local intranasal injections were instituted. The injections were given daily, and the dose was increased gradually from 5 mg. to 25 mg. She obtained a violent reaction on the tenth day, when she was given a dose of 25 mg. This injection was like all of the others except for dosage. The site chosen on each occasion was the nasal septum because it was the only visible area.

Day		Remarks
2/5/51	First day	First intranasal injection—5 mg.
	Fifth day	Dose increased to 10 mg.
	Tenth day	Dose 25 mg. Reaction within one or two minutes—weakness, dyspnea, substernal pressure, flush of entire face, wheezing, rapid weak pulse, collapse. The flushing subsided within three minutes, the wheezing within an hour.
	Eleventh day	Dose reduced to 5 mg. No reaction.
	Fifteenth day	Slight improvement in airway.
	Thirty-seventh day	Some breathing space available for the first time. Cortisone discontinued.
	Thirty-eighth day	Removal of all polyps including posterior turbinal tips
3/23/51	Forty-eighth day	Return of polypoid edema and swelling.
	Eleventh day	Dose reduced to 5 mg. No reaction.
	Fifteenth day	Slight improvement in airway.
	Thirtyseventh day	Some breathing space available for the first time. Cortisone discontinued.
	Thirtyeighth day	Removal of all polyps including posterior turbinal tips.
3/23/51	Forty-eighth day	Return of polypoid edema and swelling.

The 5 mg. doses were given up until the thirty-eighth day. The improvement was not great at that time, but she was able to breathe through her nose, and it gave me a chance to see the interior and clean out all the polyps.

The histology of all of the tissues removed before and after cortisone treatment was compared. The only possible change that I could see, and I am not sure of it, was a decrease in the intracellular edema or hydrops. The following histologic findings were present and are typical of allergic polyps in general:

1. Epithelium—increased mucus formation and hyperplasia
2. Basement membrane—hyalinization and thickening
3. Stroma—marked edema, hyperplasia and increase in the myxoid collagen tissue and cells
4. Glands—increased content of mucus; dilated and cystic ducts with plugs in them
5. Infiltration—definite tissue eosinophilia, round cell infiltrations, plasma cells and lymphocytes
6. A lymphatic follicle—in one of the polyps

Comments on ACTH and Cortisone

Obviously, cortisone is of limited value, and I think it is dangerous in the nose. In cases treated with ACTH, I have seen a chronic sinus discharge and edema subside, but not so the cysts. I have watched them with an antroscope and they are still there.

Bordley[5] was the first to use ACTH and cortisone for polyps; but he admits that the manner and site of action is obscure. The surgical removal of nasal polyps is far more practical than the injection of hormones and certainly is much safer, as stated in my discussion of the paper given a few days ago by Dr. G. W. Bissell, "New Concepts in the Use of ACTH in Ophthalmology and Otolaryngology." Regarding the severe reaction to intranasal injection of

cortisone, many explanations might be offered, but is it possible that we stimulated the pharyngeal pituitary gland?

CONCLUSION

Allergy is just one branch of modern rhinology. I have attempted to describe and illustrate as much of the pathology as time and space will allow. As stated, the present report is only one chapter. The forthcoming chapters, dealing with nasal smears and suppurative sinusitis will appear in the Manual of Applied Pathology of the Ear, Nose and Throat, which is to be published in connection with the Home Study Courses of The American Academy of Ophthalmology and Otolaryngology.

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INTRACELLULAR MORPHOLOGY OF NASAL SECRETIONS WITH KODACHROME ILLUSTRATIONS

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THE cytologic study of nasal secretions has been recognized since 1927 as an important aid in the clinical diagnosis and differentiation of certain nasal and sinus conditions. In that year, studying the nasal manifestations of allergy, Eyermann[6] reported the identification of the four main types of cells in nasal secretions. He emphasized the eosinophil and merely noted the epithelial and mononuclear cells, which we are to describe in detail. Dean[4] in 1932 stressed the significance of the nasal smear in diagnosing allergy of the nose and sinuses. Hansel[7,8] has continued to point out the importance of the eosinophil in relation to and in proportion to the neutrophilic leukocyte. These observations have been helpful in distinguishing allergic from infectious conditions or in establishing the coexistence of both. Many others including Tillotson,[17] Sewall and Hunnicut,[16] Lindsay and Walsh,[14] Kahn and Stout,[13] Johnson and Goldstein[12] have contributed to the field of nasal cytology.

This paper is concerned with the other less frequently recognized or studied cellular elements, namely the epithelial cells and the mononuclears. The Papanicolaou stain,[15] which has proved so valuable for the recognition and study of malignant cells, has been used and adapted to nasal secretions. The stain preserves and brings out details not previously seen in clinical material with Wright's, Giemsa's, or Hansel's stains. It makes possible the accurate differentiation of the types of epithelial cells exfoliated in nasal secretions. Structural changes which occur in the ciliated columnar cells during acute upper respiratory infections are shown in the slides.[1] Hilding,[9] in 1930, observed in fresh preparations that ciliated cells are exfoliated during the common cold.

The photographs were taken with a Leica camera on a "sliding focusing copying attachment" supported over the microscope. The lenses and the extension tubes were varied to give the desired magnification and size. A 95× objective and a 15× ocular were used. The magnification on the film can be estimated by comparison with an erythrocyte or leukocyte. It varies from one to four thousand times. A special light meter was calibrated for use on the ground glass.

First is shown a group of normal ciliated columnar cells taken from the human nose and prepared with the Papanicolaou fixation and stain. In the normal nose these cells are not exfoliated in any number and since there is usually very little secretion, the material was obtained by gentle wiping with a small cotton applicator moistened with saline. Certain points are noteworthy. The cilia attached to the broad end of the cell are seen distinctly, and the ciliary bodies are often discrete in the pink border. The oval nucleus has a definite thin membrane and shows a fine dispersion of chromatin with two or

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three nucleoli. It is usually located at the broad end of the cell opposite the cilia. The borders of the cell may or may not be distinct. At times the cytoplasm appears very elongated in the smears, which is evidence of its elasticity. A cytoplasmic tail is usually prominent. The methods of demonstrating human cilia in the past, except in the living condition, have been somewhat difficult. We have used supra-vital staining with neutral red. This is fairly satisfactory, but the material must be observed immediately, which is not always practical. In these slides the ciliated cells are readily distinguished from the squamous and basal epithelial cells.

We have made consecutive smears at timed intervals during the course of acute upper respiratory infections in both Papanicolaou and Wright's stains. There are now twenty-five such series. The ciliated cells are in striking contrast to the normal. It has been possible to chart the specific pattern of degeneration which the ciliated cells undergo in acute upper respiratory infections. This pattern has not been found in other types of epithelial cells nor have degenerating ciliated cells been found in allergic rhinitis unless complicated by acute infection.

Early stages of the degeneration appear in smears taken during the first two days of an infection. The chromatin, normally finely granular, is seen in larger aggregates which tend to marginate to the nuclear membrane. The cells appear shortened. A small mass of pink substance is seen among the masses of chromatin and with careful focusing it may appear to be in contact with a mass of chromatin. As the masses of chromatin agglutinate and enlarge, clear areas form and when the pyknosis is complete, there is a definite halo. Often present within the halo are small black and pink bodies. The nuclear membrane becomes quite prominent. The nucleoli have disappeared. The acidophilic cytoplasm becomes coarsely granular, shortened and widened. Smooth pink globules, small to large in size, single to several in number, tend to appear near the nucleus in the cytoplasm. The cilia remain intact. In some instances they grow from the sides of the cells.

As the degeneration progresses, the cytoplasm tends to separate from the nucleus by elongating slightly and constricting near the nucleus. Many pyknotic nuclei with clear halo often containing one or more of the small perinuclear bodies are seen separated from the cytoplasm which still retains the tuft of cilia. Throughout this obvious degeneration the cilia remain intact, even after the nuclear portion breaks off. In fresh preparations the beating of tufts of cilia without nucleus has been observed for as long as two hours. The peak of the exfoliation is from the second to the fifth day of the infection. After that the degenerating ciliated cells are difficult to find.

In the same smears the eosinophils and the neutrophils are very prominent. Eosinophils may be seen in the first smears taken from acute upper respiratory infections along with neutrophils and various types of epithelial cells. With the Papanicolaou stain the eosinophils are not brilliant nor are the granules distinct. The fixation, however, does preserve the pseudopods. Active motility of the eosinophil is easily seen in the living condition. This fixation is rapid, thus all manner of shapes are seen. They are easily distinguished from the neutrophils. The intensity of the eosinophilic color can be increased by adding a small amount of 0.5 per cent phloxine to the stain. Smears taken at the end of the second day may show so many eosinophils that they superficially resemble allergic smears. The neutrophils sometimes are outnumbered by

the eosinophils and the mononuclears. At the same time the degenerating ciliated cells are found in large numbers, but after the second day the later stages of degeneration predominate.

Mononuclears may be found in large numbers on the second day. In Wright's stain they appear to be filled with coarse blue granules so that the nucleus of the cell is often obscured. They vary in size from that of a small lymphocyte to that of a regular blood monocyte. They are often the predominant cell for a brief interval and remain numerous throughout the course of the infection. They seem to have some relationship to eosinophils. They are often found in allergic smears, sometimes in large numbers. They may be confused with lymphocytes if the stain is not unusually light. In the Papanicolaou stain their nuclear detail is very distinct. They are seen to phagocytize cell fragments.

On the fourth and fifth days all cells tend to diminish, although neutrophils, eosinophils and mononuclears are still present. Small lymphocytes may also remain. The degenerating ciliated epithelial cells are completely gone from the secretions before the other cells disappear. The cytology varies, of course, with the severity and duration of symptoms, but this general pattern of the ciliated cells has been especially constant.

The question may be asked, "Are these morphologic details really artefacts caused by some peculiarity of fixation and staining?" In order to answer this question we examined, from the same material, fresh unstained wet smears as well as those prepared with both Wright's and Papanicolaou stains. If the smears were very lightly stained with Wright's after being air dried, not flamed, and within one-half hour after being made, the same degenerating ciliated cells could be seen. They probably would not have been recognized except for the fact that they were first clearly noted in the Papanicolaou stain. The cilia are not well preserved but can be recognized. The halo and inclusions, which are of a different shade from the pyknotic nucleus, may be verified. It is also interesting to note that in the fresh specimens the same structural variations can also be observed when the lighting is indirect. The separation and splitting of the nuclear portion from the cytoplasmic portion of the ciliated cell can likewise be seen in the living condition.

For the best interpretation it seems essential to study comparatively slides made in several stains and also slides from other nasal diseases. For comparison we mention here a complete series of slides made from an attack of ragweed pollenosis lasting from August 17 to October 1, 1950. There were rather numerous squamous epithelial cells, but in all the Papanicolaou slides, only four ciliated cells were found. These did not show the type of degeneration described here. This particular case was studied with duplicate slides in Wright's stain. The dominant cell in the series was always the expected eosinophil, which tended to occur in large clumps consistent with the allergic pattern. There was always a scattering of neutrophils. On the fifth day a few small mononuclears appeared and were seen in small numbers in most of the succeeding slides. Clinically during this ragweed season the patient had no symptoms of intercurrent infection. He received no desensitization but controlled his symptoms with antihistaminics. The cytologic report is consistent with an uncomplicated attack of allergy.

Degenerating ciliated cells are often found in many of the single slides from cases of mixed allergy and infection. On the contrary they have not been found in the secretions irrigated from the sinuses in subacute and chronic sinusitis.

It follows that these cells have not been noted after the second week of an acute infection.

The abnormal nuclei of malignant cells have nothing comparable to this pattern of degeneration. Malignant cells after exposure to roentgen rays show marked degenerative changes. Time does not permit showing these here. Vesiculation, multiple nuclei, intracellular swelling, bizarre shapes and even pyknotic nuclei are common, but these alone or in combination do not simulate this pattern of degeneration.

Years ago excellent evidence of the virus etiology of acute upper respiratory infections was presented by Dochez and others.[5] Hilding[9] in studying stained and sectioned biopsy specimens of the common cold found that bacteria were never seen invading the epithelial cells. The type of degeneration presented here, we believe, is evidence in the direction of virus etiology. We wish to emphasize that these are not pictures of virus elements but that the changes are similar to those of other cells infected with virus. Dr. Edmund V. Cowdry, Dr. Walter Siebert, pathologist, and Dr. Walter P. Covell have studied these slides and concur in this opinion.

The predominant eosinophil has long been thought of as a diagnostic sign of allergy. Do not the large numbers of eosinophils in an acute upper respiratory infection suggest an allergic response? Thus the cytology as pictured here suggests factors of both virus infection and allergy in the etiology of the common cold.

It seems possible that with more detailed studies of nasal exudates at high magnification and with the use of new stains in both living and fixed material, new patterns of nasal cytology may be worked out in the future.

SUMMARY

1. Intracellular detail and cilia are demonstrated clearly by use of the Papanicolaou stain.

2. A pattern of structural changes occurring in the exfoliated ciliated epithelial cells during acute upper respiratory infections has been charted.

3. Mononuclear cells in nasal secretions have been studied and described.

4. Cytologic evidence points to the fact that in acute upper respiratory infections the location of the disease probably is within the nucleus of the cilated columnar epithelial cell.

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THE "THROW-AWAY" BUTTON-END KNIFE BLADE

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I AM presenting an interchangeable button-end knife, commonly used in rhinoplasty, with a "throw-away" blade. The blade has to be extremely sharp because of its precise work. However, due to methods of sterilization and cutting of cartilage it becomes dull very readily. The button-end knife requires almost constant sharpening and has to be replaced frequently.

For many years I have tried to make an interchangeable button-end knife similar to the principle of the Bard-Parker handles and blades. I have now constructed one in which the button-end blade can be inserted, fixed, and changed as often as desired (fig. 1). The handle, which is perfectly balanced, has a screw at the bottom which on being turned and pushed forward disengages and releases the blade. A fresh blade can be engaged by reversing the action of the screw.

The new handle with the "throw-away" button-end knife blade, in addition to its utility, will have an added advantage in its economy.

It is not unlikely that in the future the other blades so easily dulled in rhinoplastic surgery will be of the same "throw-away" type on similar handles so that they can be economically and handily changed at the will of the operator.

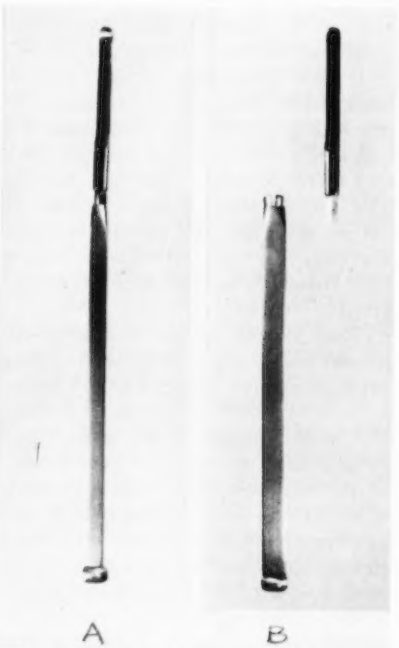

FIG. 1—Button-end knife with "throw-away" blade. (A) Assembled knife. (B) By turning and pushing the screw at the end of the handle forward, the blade is released. A fresh blade may be engaged by reversing the screw action.

From the division of rhinoplasty, department of otolaryngology, Mount Sinai Hospital, New York.
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ALLERGY OF THE INNER EAR
(MÉNIÈRE'S DISEASE)

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ALLERGY, whether it is of the immunologic or physical type, appears to be basically a localized autonomic dysfunction of the cholinergic type. This dysfunction produces a stereotyped reaction of the peripheral vascular bed (the arteriole, capillary and venule) in which arteriolar spasm is associated with dilatation of the capillary and venule that sometimes amounts to a varicosity. Müller termed this condition the "spastic-atonic state." Anoxia of the capillary loop with injury to the endothelium and contained circulating cells follows. Such anoxia results in "sludging" of the blood, local release of toxic metabolites and increased permeability of the capillary loop. The last, in turn, results in the formation of the typical allergic wheal.

When allergy affects the inner ear, this invariable reaction of the capillary bed may involve either the stria vascularis, or branches of the internal auditory artery supplying the cristae and maculae or both together. Involvement of the stria vascularis should result in an increased volume of endolymph with an increased protein content and consequently higher colloid osmotic pressure. Involvement of the branches of the internal auditory artery supplying the cristae and maculae by producing local anoxia may give rise to crises of vertigo. Involvement of both areas at once may give rise to the typical "triad" of Ménière's disease.

From the Section of Otolaryngology and Rhinology, Mayo Clinic.
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Although immunologic allergy may be the precipitating factor in Ménière's disease, physical allergy appears to be the more usual.

When the stria vascularis is involved, increased transudate with increased protein content goes into the endolymph system, producing hydrops.

Endolymphatic hydrops is considered to be primarily a distention of the cochlear duct with secondary extension to the utricle and otic sac.

Dix, Hallpike and Hood[14] in discussing the pathologic changes in Ménière's disease stated that characteristic alterations are to be seen in Corti's organ. The cell mass is compressed, the cell outlines obscured and the staining differentiation between nuclei and cytoplasm virtually extinguished. These abnormalities are so striking, according to Cawthorne,[10] that it is inevitable that they should be regarded as distinctive of Ménière's disease in certain of its phases. That they are found sometimes and not always only means that they represent a transient and reversible reaction on the part of the hair cells to the chemicophysical disturbances which go with the gross distention of the endolymph system. It seemed reasonable to Cawthorne that this change should be phasic in character, thus matching the clinical course of the disease, and also that the morphologic changes in the hair cells should vary at the same time.

Dix, Hallpike and Hood[14] tended to correlate the compressive changes found in the organ of Corti with deafness and particularly with the phenomenon of

recruitment of loudness originally described by Fowler.

Because the endolymphatic membrane is a semipermeable membrane and because ordinarily the osmotic pressures of the two humors are nearly the same, many authors have found it difficult to explain satisfactorily the pathologic finding of Hallpike and Cairns[18] that Reissner's membrane and the remainder of the endolymphatic membrane is ballooned out and the organ of Corti compressed.

A reasonable explanation appears to be that the semipermeable membrane of the endolymphatic system is relatively impermeable to protein molecules. Therefore, the Donnan effect would be relatively large in such a situation since it is directly proportional to the concentration of the nondialysable substance. This would result in increased osmotic pressure in the endolymph system. The solvent water would then be drawn into the system from the perilymph and distention of the membrane with increased pressure on the organ of Corti would result.

Arnvig[4] and Berggren[8] reported apparently authentic cases of Ménière's disease without hydrops in which no alterations could be found in the labyrinth. Brunner[9] reviewed the literature on the pathologic changes of Ménière's disease and stated that in some of his own cases in which the patients had suffered from recurring dizzy spells, hydrolabyrinth was not found at necropsy. He cited Wittmaack to the effect that hydrolabyrinth may consist of distention of the membranous labyrinth owing to hypersecretion of endolymph. He stated that serous labyrinthitis or hydrops will not invariably cause hydrolabyrinth and that hydrolabyrinth may be a reversible process, although it eventually becomes irreversible. By hydrolabyrinth Brunner[9] apparently means an increase of fluid and protein material in both the endolymphatic and perilymphatic spaces, while by serous labyrinthitis he appears to indicate an increase in fluid without a concomitant increase, or possibly even a reduction, in its protein content.

Brunner[9] stated that Ménière's disease was essentially vasomotor labyrinthitis. He argued that vasomotor labyrinthitis may or may not cause the symptoms of both vertigo and deafness. He stated that the histologic findings of an accumulation of albuminous fluid in the inner ear are not invariably associated with cochlear plus labyrinthine symptoms, regardless of whether the abnormal permeability of the blood vessels is inflammatory in origin or not. His investigations and reasoning left Brunner at a loss for an explanation of the facts he had observed. Much of his difficulty in forming a reasonable hypothesis to explain the divorcement of the cochlear and vestibular symptoms in Ménière's disease seems to have been due to two factors: 1. His criteria for the diagnosis of Ménière's disease differ somewhat widely from those of Ménière and many other observers. 2. In considering the pathologic basis of Ménière's disease, he concentrated his observations exclusively on the postmortem histopathology and ignored the lessons of physiopathology which have long been pointed out by observers using the technic of biomicroscopy.

Békésy[7] cast doubt on the hypothesis presented by Dix, Hallpike and Hood,[14] and Cawthorne[10] that compression of the cells of the organ of Corti produced by increased pressure in the endolymph would produce the characteristic changes of hearing found in endolymphatic hydrops. He was unable to show experimentally that increasing the pressure of the perilymph and endolymph produced a reduction in hearing. Lempert and his associates[22] in confirming Békésy's work stated that their observa-

tions showed in addition that increase of pressure in the humors did not interfere with the functioning of the sensory cells of the organ of Corti under the conditions of their experiments.

The evidence secured from the human being with endolymphatic hydrops appears to be directly opposed to that presented by Békésy from experiments on fresh cadavers and by Lempert and his associates[22] from experiments on the monkey.

Altmann and Waltner[3] called attention to the fact that the fluid exchange in the inner ear takes place through semipermeable membranes and that the fluids involved are not simple, true solutions as in the experiments. The weight of evidence now favors the position that the conditions of the experiments done by Lempert and his associates do not reproduce the conditions found in the inner ear in Ménière's disease and that therefore their evidence may be misleading.[39]

In times past, the deafness of Ménière's disease has been referred to as being of the nerve type. Mygind[27] indicated that this statement was in error. He suggested that the deafness, while of an inner ear type, was in all probability due to changes in the "humors" of the inner ear and, therefore, was due to interference with conduction in the inner ear.

Wright[43] pointed out that the deafness of Ménière's disease may precede any other sign of the disorder by months or years. He termed this type of deafness "cochlear deafness" to distinguish it from the loss of hearing seen in degeneration of the fibers of the auditory nerve or spiral ganglion.

A characteristic feature of the deafness, at least in the early stages of Ménière's disease, is its tendency to increase and decrease in a seemingly capricious manner. As shown by Lillie, Horton and Thornell,[24] ability to hear may vary within a range of from 10 to 30 db. (decibels) in the course of several days. These changes are not abrupt, however, and the fluctuations in hearing may be entirely independent of the presence or absence of vertigo. Dederding[12] showed that this "cochlear" deafness of patients who have Ménière's disease can be influenced by the intake of salt and fluid. Her evidence indicated, also, she stated, that increasing the intake of sodium and fluid could precipitate the vertiginous crisis of Ménière's disease. Furstenberg, Richardson and Lathrop[16] extended the investigations of Dederding and demonstrated that increasing the intake of sodium alone would precipitate a vertiginous crisis of Ménière's disease, whereas increasing the intake of fluid and restricting the intake of sodium would not precipitate one. They concluded that it was the excess of sodium itself, not "water loading" secondary to the ingestion of sodium as Dederding stated, that precipitated the vertiginous crisis in this experiment.

The evidence furnished by Stoesser and Cook[36] and Kern[20] indicates that an excessive intake of sodium lowers the threshold for allergic reaction. It seems possible that the precipitation of the vertiginous crises of Ménière's disease in these so-called water-loading experiments, which was presumed to have been due to increased pressure in the endolymph, may have in fact been precipitated by an excess of the sodium ion producing vasospasm in the branches of the internal auditory artery.

More than forty years ago Shambaugh[33] suggested the hypothesis that the recurring vertiginous crises of Ménière's disease were produced by vasospasm. He pointed out that due to the anatomic arrangement, disturbance of the circulation to the labyrinth may be limited to one or several of the terminal arterioles supplying certain areas without involving the remainder of the labyrinth.

Lermoyez[23] elaborated the hypothesis that circulatory dysfunction was the precipitating cause of the crisis. He suggested that deafness, tinnitus and vertigo might result from either dilatation or constriction of the vessels supplying the labyrinth. He pointed out that a spasm of the internal auditory artery would affect simultaneously the equilibrial labyrinth and the cochlea, which would manifest their trouble by the complete symptomatic triad of Ménière. Spasm of the branches of the vestibule on the other hand might precipitate paroxysmal vertigo without concomitant effect on the hearing, whereas spasm of the cochlear branches might provoke deafness and tinnitus but no vertigo. Since this time most students of Ménière's disease have considered it a vasomotor labyrinthitis or, as it was recently put by Hilger,[19] vasomotor labyrinthine ischemia.

If the cochlear changes in Ménière's disease are considered due to the stereotyped vascular change of allergic autonomic dysfunction taking place in the stria vascularis, with a secondary increased transudation of the proteins of the serum, while the vertigo is on the basis of the same type of dysfunction of the peripheral vascular bed affecting the blood supply to the cristae and maculae and producing immediate stimulation by anoxia, a separation of phenomena which is definitely possible on an anatomic basis, there appears no particular reason why endolymphatic hydrops both with and without vertigo should not occur. Ménière's disease, on the same basis, can be conceived of as occurring with and without endolymphatic hydrops.

The problem of allergy of the inner ear may be made more easily comprehensible by a brief review of the humoral systems and the blood and autonomic nerve supply to the labyrinth.

ANATOMIC AND PHYSIOLOGIC FACTORS

The Humoral System of the Labyrinth

The humoral system of the labyrinth is made up of a completely closed system, the endolymph system, surrounded by a second system, the perilymph system, which in laboratory animals communicates with the arachnoid sac through the aquaeductus cochleae (perilymphatic duct). These two systems are separated for the most part by a semipermeable, flat endothelial membrane except where separation is produced by the basilar membrane. In man under normal conditions, according to Aldred, Hallpike and Ledoux,[1] the osmotic pressure of the endolymph is slightly greater than that of the perilymph.

Altmann and Fowler[2] stated that although many authors assume that pressure equilibrium between the cerebrospinal fluid and the perilymph is maintained by the cochlear aqueduct in human beings as well as in animals, this is not necessarily so. They pointed out that Meuerman found this aqueduct in man to be relatively much narrower than in experimental animals and ordinarily filled with dense connective tissue and often with bone. Zange[44] was struck with the narrowness of the cochlear aqueduct and compared it to a curved capillary tube. He found that the viscosity of the perilymph and of the cerebrospinal fluid was high and concluded that this would prevent sudden sharp rises in endolymphatic and perilymphatic pressure from being quickly transmitted to the subarachnoid space even if the cochlear aqueduct were open. Waltner's findings[38] suggested that in the rabbit there is an endothelial membrane at the junction of the vestibule and the cochlear aqueduct. In his opinion this showed that even in the rabbit an increase in volume and pressure of viscous fluids such as the humors of the inner

ear probably would not be relieved by "free communication" with the arachnoid sac. Such communication might be obtained only in exceptional circumstances with rupture of the occluding membrane of the cochlear duct.

The recent work of Bast and Anson[5] appears to reassert the hypothesis that most of the endolymph is carried off through the otic sac. Most observers are of the opinion that perilymph originates from endolymph by transudation through the endolymphatic membrane. The osmotic pressures of the humors of the inner ear have been found to be definitely higher than the osmotic pressure of the cerebrospinal fluid. The slight difference in osmotic pressure between the endolymph and perilymph may be due to the Donnan effect, since the endolymph may be presumed to have a higher concentration of protein because of the possibility for the increased transudation of protein particles into the endolymph through the stria vascularis.

The organ of Corti receives all its nutrition from the endolymph, since no blood vessels supply it. For this reason it differs from other organs that receive vascular supplies. This difference, however, is more apparent than real because all cells receive their nutrition from lymph. Anatomists have recognized that the humoral systems of the labyrinth are analogous and comparable to an enlarged lymph space by their nomenclature, "perilymph" and "endolymph." The difference between the humoral systems and other lymph spaces lies in the greater size of the former. This factor, however, may be expected to have definite effects on the rapidity with which osmotic and chemical adjustments take place in the humors.

Since the work of Guild,[17] the stria vascularis has been generally accepted as the source of endolymph. Shambaugh,[34] however, described certain glands in the sulcus externus which he considered to be a partial source of the endolymph. Wittmaack[41] stated that the endolymph was formed from a transudate from the vessels of the stria vascularis and a secretion from glands, the former rich in protein, the latter similar to a saline solution. Wakim[37] considered this a reasonable assumption because physiologists are beginning to consider that all serous fluids of the body are a combination of a transudate from the capillaries and of glandular secretion. This combination would offer a mechanism by which compensation for changes in the osmotic equilibrium could be rapidly effected.

Guild[17] found that the endolymph in the cochlear duct moves toward the basal end and passes through the ductus reuniens into the otic sac (endolymphatic sac). He concluded that the endolymph passes through the wall of that part of the otic sac which he termed the "pars intermedia." In his opinion structural differentiation indicated that this is a functionally specialized region. This opinion has recently been substantiated by Bast and Anson[5] and by Arnvig.[4] Bast and Anson answered some of the objections proposed by McNally[26] and Lindsay[25] to Guild's hypothesis.[17]

Saxén[31] and Kobrak[21] suggested that the endolymph was secreted by the cells of the stria vascularis and resorbed by the cells of the sulcus externus. Ruëdi[30] made some observations which he thought pointed to this conclusion. The weight of the evidence, however, appears to favor the hypothesis originally presented by Guild.[17]

In the opinion of Altmann and Waltner,[3] the fact that the endolymph and perilymph are practically iso-osmotic favors the assumption that the endolymph gives origin to at least part of the perilymph. They also stated that morphologic evidence suggested that the modiolar vessels might be a possible

site of origin of part of the perilymph, especially in man and monkeys.

The Elimination of the Labyrinthine Humors

As mentioned before, the endolymph and perilymph systems appear to be analogous to lymph spaces in other portions of the body. Elsewhere in the body interstitial fluid tends to be eliminated *via* the lymph vessels. Movement of lymph generally is promoted by the contraction and relaxation of muscles and by vascular pulsation. In the inner ear, however, nature appears to have eliminated the possibility that such mechanical factors will aid in the elimination of interstitial fluid.

Pulsations of the internal carotid artery are largely eliminated when it passes through the carotid canal, as is indicated by the fact that under normal conditions the arteries of the retina do not pulsate. Thus reduction of all pulsation occurs within the cranium. The long narrow labyrinthine artery (or the antero-inferior cerebellar artery, whichever nomenclature is accepted) tends to diminish arterial pulsation in the inner ear. In addition Mygind[27] has described certain "glomeruli" in the arterial loops to the cochlea which tend to eliminate pulsations and other changes in pressure. Pulsations in the brain are prevented from affecting the humors of the inner ear by the long, narrow vestibular aqueduct designed physically to absorb and dampen any waves of alternating pressure transmitted from the brain and cerebrospinal fluid. Pulsations in the cerebrospinal fluid also are prevented from affecting the humors by the fact that in man the cochlear aqueduct (perilymphatic duct) does not appear to communicate with the arachnoid sac.

Therefore actual secretion, as suggested by Bast and Anson,[5] and the hydrostatic and colloidal osmotic pressures, as suggested by Altmann and Waltner,[3] seem to be the factors that must be largely effective in the elimination of an increased endolymph of higher osmotic pressure such as may be presumed to be present in Ménière's disease with endolymphatic hydrops.

Autonomic Nerve Supply to the Labyrinth

The weight of the evidence favors the opinion that the adrenergic (sympathetic, thoracolumbar) nerve supply to the labyrinth is along the vertebral, basilar and labyrinthine arteries.

Rasmussen[29] suggested that the cholinergic (parasympathetic, craniosacral) nerve fibers to the labyrinth originate in the superior olive and travel by the olivocochlear fasciculus to the vestibular branch of the eighth cranial nerve, reaching the cochlea via Oort's vestibulocochlear anastomosis. It has not been conclusively settled whether vasoconstriction in the labyrinth is produced by craniosacral or thoracolumbar impulses. Experimental evidence seems to favor the latter conclusion slightly, but from the clinical standpoint the former seems the more reasonable.

The Vascular Supply to the Labyrinth

The arterial supply to the labyrinth appears to be from a long narrow vessel termed by Siebenmann[35] the "internal auditory artery" and by Nabeya[28] the "antero-inferior cerebellar artery" which divides into the vestibular artery, the vestibulocochlear artery and the cochlear propria artery.

Comment. Even from this superficial résumé of the anatomy and physiology of the humoral system it can be readily concluded that several functional and pathologic changes may effect an increase in the fluid pressure of the endolymph and perilymph. To produce changes in the humors, the secretory mechanism, the absorbing mechanism or

the osmotic pressure relationships would have to be involved.

The relative slowness with which changes in the cochlear signs take place in Ménière's disease as opposed to the sudden onset and departure of the signs of disturbance of the equilibrial labyrinth indicates some difference in the mechanism producing these two different symptom complexes. Vasospasm producing anoxia, or sudden release of vasospasm producing irritation by a sudden resumption of blood flow, seems to explain the disturbances of the equilibrial labyrinth. In the cochlea, however, while the disorder may be initiated by changes in the stria vascularis, the relatively slow compensatory changes in the humors, as reflected by changes in hearing, must result from the slower changes in osmotic equilibrium initiated by changes in the stria vascularis and their effect on the composition of the endolymph. The evidence available points to involvement of the peripheral vascular bed in infection, chronic degenerative states, thrombosis, embolism and allergy as the usual causes of hydrops involving the endolymph. In all these disorders except that produced by the localized autonomic dysfunction of allergy the resulting hydrops appears to be irreversible.

Factors such as "water loading," increased sodium intake and the like may produce hydrops in a predisposed labyrinth, by which is meant a labyrinth already involved in an allergic mechanism.

THE DIFFERENTIAL DIAGNOSIS OF MENIERE'S DISEASE AND ENDOLYMPHATIC HYDROPS

Good therapeutic results in the allergies of the inner ear depend largely on their careful distinction from conditions which may simulate allergies and yet cannot be reversed by the therapeutic methods effective in the allergies.

Several cases illustrating some of the difficulties and pitfalls of differential diagnosis will be given.

Although the usual description of endolymphatic hydrops on an allergic basis suggests that differentiation from other types of deafness should prove comparatively easy, in my own experience this has not always proved to be so. Deafness due to endolymphatic hydrops is frequently described as perceptive in type and as readily separable from deafness due to otosclerosis or from other conduction types of deafness by the Rinné test. Results of this test are said to be always positive in perception deafness and negative in conduction deafness.

Although this usually holds, exceptions are occasionally met. Dix, Hallpike and Hood[14] pointed out that occasionally in the deafness of Ménière's disease the Rinné test gave a negative result even when absolute bone conduction was tested for. This has been my experience also. I believe it is due to two factors: (1) patients who are completely unable to differentiate sound and vibration, and (2) an even smaller group of patients in whom a negative result of the Rinné test is actually obtained in endolymphatic hydrops. Dix and Hallpike[13] suggested that the negative result in the latter group is owing to a considerable increase in the density of the labyrinthine fluid, if not to the formation of an actual coagulum. When a negative Rinné test is associated with a history of slowly progressive deafness over many years, accentuated by pregnancy, the differentiation from otosclerosis is not at once obvious. This type of case, however, is usually not allergic in origin and, therefore, probably should not be classified as Ménière's disease. The following patient presented this difficulty in diagnosis:

Case 1. A white woman, 36 years of age, stated that she had first noticed deafness at

the age of 13 years. There was not much further change in hearing until she noticed a considerable progress in her deafness with the birth of each of her two children. She had never had any tinnitus, vertigo or nausea. She felt that she heard better in a noisy place than in a quiet one, but felt she heard women's voices better than men's. She had worn a hearing aid for more than five years but did not think that it improved her ability to hear in the slightest degree.

The Rinné test with tuning forks gave negative results in both ears at 256 cycles per second. This patient had been referred to me for consideration for the fenestration operation. General and laboratory examinations gave essentially negative results except for the hearing tests. Threshold audiograms for pure tones and discrimination test for phonetically balanced words were obtained (fig. 1).

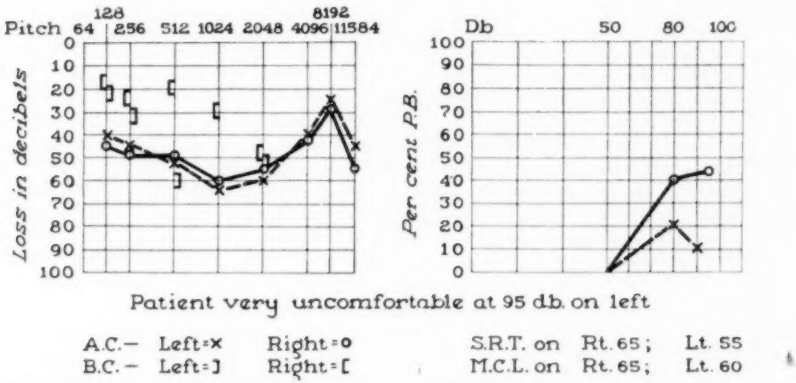
Pitch 64 128 256 512 1024 2048 4096 8192 11584
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
Db 50 80 100
Per cent P.B.
100 90 80 70 60 50 40 30 20 10 0
Patient very uncomfortable at 95 db. on left
A.C.— Left=× Right=o
B.C.— Left=] Right=[
S.R.T. on Rt. 65; Lt. 55
M.C.L. on Rt. 65; Lt. 60

FIG. 1—(Case 1) These audiometric findings appear to indicate the presence of endolymphatic hydrops. The patient, a woman 36 years of age, was referred for a fenestration operation because of negative results of the Rinné test.

These findings seemed to indicate endolymphatic hydrops. While a loudness balance test could not be done, it was felt that the minimal interval between the speech reception threshold and the level of most comfortable loudness in both ears as well as the discrimination deafness indicated the presence of endolymphatic hydrops.

It might be argued that endolymphatic hydrops of allergic type had occurred secondarily in two ears which were already the seat of otosclerosis, but nothing in the history points to the onset of such a condition. This patient had gone 23 years since the onset of her deafness without either vertigo or dizziness. I subscribed to the hypothesis that the loss of hearing was on the basis of an endolymphatic hydrops from the first, especially since I had seen several somewhat similar cases. The essentially vascular histopathologic change shown by Dorothy Wolff[42] to be present in otosclerosis seems to make the association of otosclerosis with endolymphatic hydrops a defensible suggestion, however. This type of endolymphatic hydrops has been found to be completely irreversible under treatment so that the fundamental physiologic mechanisms of its production may be different from those of the reversible variety. I have also found, however, that severe deterioration of discrimination for phonetically balanced words may not always be present in what appears to be a definite instance of endolymphatic hydrops not on an allergic basis. The following case appears to illustrate this point.

Case 2. A man, aged 43 years and weighing 252 pounds (114.3 kg.), who had reduced his weight from more than 350 pounds (158.7 kg.) in the previous year, stated that he had had one severe attack of vertigo, nausea and vomiting in each of the years, 1937, 1938, 1939,

TABLE I

EQUILIBRATION TEST*

CANALS STIMULATED	ICE WATER, CC.	NYSTAGMUS				VERTIGO
		TYPE	DEGREE	AMPLITUDE	DURATION	
Right horizontal	5	→	2	2	55″	Mild
	10	→	2	2	1′	
	30	→	2	2	1′25″	
Left horizontal	5	←	3	2	1′40″	Mod.

*No spontaneous nystagmus or positional nystagmus; hypoactive labyrinthine response on left.

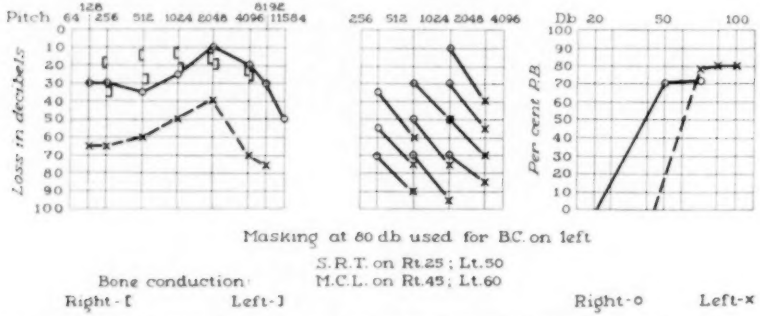
Pitch 64 128 256 512 1024 2048 4096 8192 11584
Loss in decibels
256 512 1024 2048 4096
Db 20 50 100
Per cent P.B
Masking at 80 db used for BC. on left
S.R.T. on Rt.25 ; Lt.50
M.C.L. on Rt.45 ; Lt.60
Bone conduction:
Right-[Left-]
Right-o Left-×

FIG. 2. (Case 2) Probable diabetic vasculitis with bilateral endolymphatic hydrops. Endolymphatic hydrops is indicated by the narrow "dynamic range" in the two ears (the difference between the speech reception threshold and the minimal comfort level). The lack of evidence of recruitment shown on the loudness balance test is thought to be owing to the presence of hydrops on both sides. The lack of discrimination deafness present seems to indicate an absence of compression of the organ of Corti.

1945 and in 1949. In the spring of 1950 (eleven months before being seen at the Mayo Clinic) he had ten severe attacks in ten days. At that time severe tinnitus which was somewhat more marked on the left developed and hearing diminished in both ears. Since that time two weeks had been the longest period he had passed through without severe attacks of vertigo, nausea and vomiting. At the start of the severe attacks he had been given priscoline (2-benzyl-4, 5-amidazoline hydrochloride) which seemed to give him partial relief for a time. He then was given nicotinic acid in doses up to 500 mg. three times a day. This produced little flushing and no symptomatic relief. He was then given a diet containing 0.2 gm. of sodium or less and was given daily intravenous injections of 0.2 per cent solution of procaine with epinephrine and ascorbic acid for two weeks without effect. Next Jacobson's solution and then six injections of ACTH were tried without effect. Finally dimenhydrinate (dramamine), 50 mg. every three hours, together with ¼ grain (0.05 gm.) of phenobarbital every three hours was tried. He obtained more marked relief from this treatment than from any of the other medicaments; the attacks became rather mild and lasted one to ten minutes. They continued to be frequent, however, and he was completely incapacitated for his usual occupations.

General physical examination was without particular significance except for the finding of glycosuria, grade 3, on a grading basis of 1 to 4, and 195 mg. of sugar per 100 cc. of blood. The diabetes was not severe and it was thought that it would become milder with further loss of weight. A 1,000 calorie diet was recommended and he was advised to reduce his weight to 175 pounds (79.4 kg.). With this diet and without insulin his urine became sugar free.

Examination of the central nervous system, which included roentgenograms of the head with Stenver's projection to show the internal auditory meatuses, was negative.

Examination of the ears, nose and throat gave negative results except for the audiometric findings (fig. 2 and table 1). The speech reception threshold in the right ear was 25 db., and in the left 50. The level of most comfortable loudness in the right ear was 45 db., in the left 60.

In spite of the somewhat equivocal audiometric findings, a diagnosis of Ménière's disease with endolymphatic hydrops was made. The patient was given histamine intravenously without any observable effect. The diagnosis was changed later to endolymphatic hydrops on the basis of diabetic vasculitis.

The lack of recruitment in the loudness balance test in case 2 may be misleading. If hydrops is present on both sides, recruitment may not be obtainable in my experience. This is borne out by the narrow interval found between the speech reception threshold and the most comfortable loudness for speech on both sides. In spite of this, however, the discrimination curve for phonetically balanced words rises to 65 and 70 per cent on both sides.

It may appear that I was in error in classifying this as a case of Ménière's disease. It is well known that vascular changes are common in diabetes and that angina and claudication are not infrequently present. It is probable that the dysfunction of the peripheral vascular bed here depended on diabetic changes in the endothelium rather than autonomic imbalance. Certainly drugs directed toward correcting autonomic imbalance proved ineffective. Dramamine and phenobarbital, which appear to have a similar sedative action on higher centers, gave some measure of relief.

Case 3. The patient, a musician aged 27 years, stated that she had had a hearing loss in the left ear since she had measles in childhood. She felt that there was a difference of about a quarter tone in the perception of a given pure tone as heard in the two ears. She had never noted any variation in her hearing and had never experienced vertigo, dizziness or unsteadiness.

Ear, nose and throat examination gave negative results except for the hearing tests. Diplacusis was evident in the right ear as compared to the left in the following frequencies: at 128 cycles per second higher, at 256 equal, at 512, 1,024 and 2,048 lower.

Pure tone threshold audiogram showed normal hearing in the right ear. On the left the findings in decibels for air conducted sounds at various cycles per second were as follows:

128	256	512	1,024	2,048	4,096
30	30	25	30	40	35

Bone conduction was close to the 30 db. level at all frequencies in the left ear.

Loudness balance tests showed marked recruitment of the left ear over the right. Hyperrecruitment was present at frequencies of 512 and 1,024 cycles per second and recruitment was complete at the frequency of 2,048 cycles per second.

It was felt that the hearing loss in this ear in all probability represented a permanent irreversible hydrolabyrinth resulting from an acute labyrinthitis associated with measles in childhood.

Case 4. The patient, a man 38 years of age, presented himself to us because of attacks of vertigo with occasional nausea and vomiting and increasing deafness which had first appeared two years previously. The first attack began with dullness in the right ear while the patient was driving his car. Two hours later his surroundings suddenly appeared to be spinning in a counterclockwise direction. He stopped his car, got out and tried to walk but could not keep his balance. The attack lasted about two hours. He had six somewhat similar attacks in the succeeding two years with vomiting on three occasions. During the attacks hearing in the right ear decreased but returned to normal after the attacks until four months before his admission when he began to notice intermittent tinnitus with gradually decreasing hearing on the right.

During the year before admission he took ammonium chloride tablets when he felt an attack coming on, with, he believed, some symptomatic relief. He had also had a submucous resection performed a year before his admission but with no relief to his symptoms.

The ear, nose and throat examination was negative except for the findings relative to the functions of the inner ear.

The threshold audiogram for pure tones showed normal findings on the left. On the right the findings in decibels at various frequencies were as follows:

	128	256	512	1,024	2,048	4,096
A. C.	65	60	65	65	50	20
B. C.	0	60	45	30	35	

Loudness balance tests showed an incomplete recruitment of loudness in the right ear over the left at frequencies of 512, 1,024 and 2,048 cycles per second.

Testing of the equilibrial function of the labyrinths by a modification of the Kobrak test showed relatively decreased equilibrial function on the right. Examination of the eyes revealed normal pupils, reflexes and fundi. A roentgenogram of the skull, however, showed evidence of Paget's disease with osteoporosis circumscripta especially involving the left frontoparietal region and platybasia.

It has been recognized for a long time that Paget's disease of the skull may be associated with hydrops and recurring attacks of vertigo that are not reversible under treatment. Nevertheless this patient was given our standard treatment for Ménière's disease with labyrinthine hydrops under the forlorn hope that in this instance the hydrops might be an intercurrent disorder. However, after a month's trial no benefit was secured. The patient was then given dramamine, 50 mg. three times a day, which also failed to give relief.

Case 5. Ten years before his admission to the clinic the patient, a man aged 61 years, noticed while driving his car that the road seemed to be coming up into his face. He stopped the car, got out and vomited. On getting back in he was unable to drive. He was unable to walk for three days and had to be led back and forth to work for the next three months. He has never been entirely free from a sensation of unsteadiness and dizziness, but this symptom waxed and waned and was likely to be more pronounced on rising in the morning.

A month after the onset of his complaint he noticed a throbbing in his right ear synchronous with his pulse. Excitement or effort tended to make this throbbing more pronounced.

Ménière's syndrome was diagnosed elsewhere but treatment had not given any symptomatic relief. Treatment consisted of low salt diet, intravenous injections of histamine and tubal insufflations. He had little disturbance in hearing but at times he could not hear a watch tick (he was a watchmaker), and at other times he heard a watch tick better in one ear than in the other. He felt that his symptoms were distinctly less pronounced at the time of his admission than they had been ten years previously.

Ear, nose and throat examinations gave essentially negative results except for the findings in regard to the function of the inner ear.

Audiometric tests with pure tones at threshold gave the following findings in decibels:

	128	256	512	1,024	2,048	4,096
A.C. Rt.	15	15	15	10	25	40
A.C. Lt.	5	0	0	0	10	10
B.C. Rt.		15	15	15	25	
B.C. Lt.		0	50	35	40	

Recruitment was not present. The patient had a positional vertigo without nystagmus on changing from the supine to the sitting position. Tests of function of the equilibrial labyrinth by a modification of the Kobrak test with 5 cc. of ice water produced nystagmus of normal duration, direction and amplitude on both sides, but vertigo could not be elicited even when the amount of the ice water used was increased to 30 cc. In other words, there appeared to be a dissociation of vertigo and nystagmus which, according to Leidler, is produced by a lesion in the substantia reticularis.

On examination of the eyes, the pupils and reflexes were found to be normal and the fundus and fields negative. Roentgenograms of the skull including the Stenver and Towne projections showed nothing abnormal and general physical examination gave negative results except for questionable mild anemia. Except for the ear findings, examination of the central nervous system was objectively normal. It was felt that the diagnosis of a non-neoplastic lesion of the brain stem, possibly a small thrombus with infarction, would fit in well with the history and findings. The patient was given dramamine, 50 mg. three times a day, without producing any effect on his symptoms.

Although it is true that the symptoms and signs in this case are not much like those of Ménière's disease, the patient nevertheless was treated for "Ménière's syndrome" by several physicians. This should serve as a warning to us not to confuse ourselves by resorting to this meaningless diagnostic term but rather to attempt differential diagnosis.

In studying a group of approximately 500 cases of Ménière's disease with endolymphatic hydrops I felt that the finding of vestibular hypofunction in the affected ear was a sign of significance. This was particularly true in cases of cochlear deafness. However, unilateral vestibular hypofunction is so frequently found in angle lesions that testing the equilibrial mechanism is not of marked value in the differential diagnosis.

Onset of cochlear deafness is usually slow and insidious. Nevertheless, Williams, Horton and Day[40] found instances of precipitous onset and recovery and other instances in which sudden onset was not followed by remission. At that time they classified the latter as instances of thrombosis or embolism. In the next case, sudden onset of deafness was associated with recruitment and yet vigorous therapy failed to produce any change.

Case 6. This patient, a white woman aged 23 years, had noticed slight deafness in the left ear during the winter, five years before her present admission. This had appeared with no obvious cause and disappeared the following summer. She had noticed no further trouble until six weeks before coming to the clinic, when she suddenly found she could not hear from her left ear. The patient was a school teacher in the primary grades so it may be presumed that the loss of hearing appeared as suddenly as she assumed.

General physical examination, including examination of the fundus, roentgenogram of the head with Stenver's projection to show the internal auditory meatuses, and Wassermann test for syphilis, gave negative results. Tests of hearing disclosed marked diplacusis binauralis dysharmonica at 128 and 256 cycles per second and slight diplacusis at 512 cycles. No diplacusis could be made out at 1,024 cycles. The tuning forks were heard as of higher pitch in the left ear.

Caloric testing of the function of the equilibrial labyrinth by a modification of the Kobrak method gave normal findings on both sides. The speech reception threshold on the right was 0, on the left 50 db.; the most comfortable loudness of speech on the right was 45 db., and on the left 70. In the right ear 98 per cent of phonetically balanced words were received at 45 and 60 db. In the left 72 per cent of the words were received at 70 db. and 60 per cent were received at 85 db. Hearing of pure tones in the right ear was normal both by bone and air conduction. Decibel levels at which various pure tones were heard in the left ear by air conduction are as follows:

128	256	512	1,024	2,048	4,096	8,192	11,584
30	40	45	50	45	45	65	65

With masking at 70 db. bone conduction was found in the left ear at 40 db. for frequencies of 256 cycles per second and at 60 db. at 2,048 cycles only.

Loudness balance tests showed slight partial recruitment at frequencies of 500 and 1,000 cycles. At 2,000 cycles slight recruitment was present until 70 db. was reached, when complete recruitment suddenly appeared. At 80 db., however, there was again a 15 db. difference between the two ears. This finding was rechecked several times and has proved difficult to explain.

In spite of these somewhat equivocal findings the patient was given treatment for a presumed endolymphatic hydrops for two months but failed to show any change in symptoms or signs.

The only conditions in which it is suggested that hydrops may be produced by interference with absorption of the humors are the early stages of neuromas of the eighth nerve and angle tumors. In these conditions choking of the labyrinth has been said to originate from interference with the venous outflow.

Cases of this type are rarely recognized early enough so that the findings of recruitment suggesting a labyrinthine hydrops predominate over the signs produced by degeneration of the nerve

cells and fibers of the eighth nerve. However, case 7 well illustrates the phenomenon of recruitment in an angle lesion.

Case 7. A man, aged 38 years, stated that four years before admission to the clinic he became aware of drawing movements of the right side of the face, wormlike in speed and behavior. They were most marked in the chin, which became deeply dimpled. Two years before admission he noted progressive weakness of the right half of the face, blurred vision on looking toward the left, decreased hearing on the right and occasional intense pain in the base of the skull on rising from a stooping position or on sudden turning.

A year before admission he began to have daily parietal headaches on either the right or left side. Sometimes these would last all day and were of sufficient severity to make him turn pale, break out into a cold sweat and grasp his head in both hands. At such times he might not be able to see well on looking toward the left. For the month before admission, however, the headaches had been less troublesome.

Ear, nose and throat examination revealed the velum of the palate to be higher on the right than on the left but was negative otherwise except for findings in regard to function of the right inner ear. Hearing for pure tones at threshold was normal in the left ear. On the right, however, testing at various frequencies gave the following findings in decibels:

	128	256	512	1,024	2,048	4,096
A.C.	25	25	30	35	20	20
B.C.		25	35	35	20	25

The loudness balance test revealed slight and incomplete recruitment at frequencies of 512, 1,024 and 2,048 cycles per second. The narrowed dynamic range for hearing on the right was demonstrated by the 25 db. difference between the speech reception threshold and maximal comfort level on the right as compared to 45 db. on the left.

The discrimination test for phonetically balanced words, using the Central Institute for the Deaf recordings, showed a 95 per cent discrimination on the right at 50 db. whereas on the left at 15 db. above threshold a maximal discrimination of 65 per cent was obtained, which dropped to 55 per cent at a further increase of 10 db.

At operation an astrocytoma, grade 2, was found which had invaded the right internal auditory meatus from the cerebellopontine angle.

When greater pressure had been caused by firmer and larger growths, Eby and Williams found even partial recruitment to be absent.

Comment. There are many difficulties and pitfalls in the diagnosis of Ménière's disease without hydrops, Ménière's disease with endolymphatic hydrops, and cochlear deafness or endolymphatic hydrops without vertigo. Cases 1 to 7 indicate some of the difficulties that may be experienced. There is some indication that these exceptional cases may have similar end phenomena to those of Ménière's disease but resulting from a somewhat different basic physiologic mechanism. Perhaps some term ought to be found for this group other than endolymphatic hydrops, such as the "hydrolabyrinth" suggested by Brunner.[9]

In the usual cases of endolymphatic hydrops differentiation is rather readily made, as Dix, Hallpike and Hood[14] showed by the loudness balance test and by the discrimination test for phonetically balanced words. This finding has recently been confirmed by Eby and Williams.[15] In such a case symptoms are unilateral to a considerable extent at least, although Cawthorne, Fitzgerald and Hallpike[11] were able to demonstrate slight cochlear changes in more than 80 per cent of cases of Ménière's disease in the better or "unaffected" ear. It is my opinion, therefore, that the loudness balance test and the percentage reception curve for phonetically balanced words are the best guides and that when hydrops of any degree is actually present, hypoactivity in the involved equilibrial labyrinth will be found.

TREATMENT

If a definite diagnosis of Ménière's disease can be made, not only relief of

the vertiginous crises but restoration of hearing can usually be secured if the patient can and will adhere to the regimen described. Many of these patients are subject to anxiety and tension states. Relief of these conditions is of great importance in a favorable clinical result.

Since Ménière's disease appears to be an expression of an inherited constitutional tendency, it would be expecting too much of any therapeutic plan to eradicate such a tendency. Therefore even after symptoms have been relieved, recurrences are to be expected. Few patients are so situated that they can devote their entire time to control of this disorder. Treatment, therefore, should be designed with the well-being and maximal convenience of the patient uppermost in mind.

Successful medical treatment of Ménière's disease has been based on the hypothesis that the condition is due to a localized dysfunction of the normal autonomic defense mechanism which includes the peripheral vascular bed, in other words an allergy. So-called specific therapy, such as elimination of foods and contacts or specific desensitization, however, has successfully relieved the disorder in a few cases. Such immunologic reactions may be a continually recurring precipitating factor and the possibility of their presence should not be ignored. In my experience, however, the more frequent precipitating factor is a physical stimulus.

Therefore, use of a logical combination of therapeutic agents known to affect the various factors operating in physical allergy seems reasonable, rather than use of one and then another agent in tandem. As Williams, Horton and Day[40] reported in a previous paper, the first measurable evidence of the restoration of hearing may not appear until after two to four weeks of vigorous treatment. Therefore, therapeutic trial should not be given up too early.

Vasodilators. The use of vasodilators to relieve the arteriolar spasm present in allergy is one of the first methods of treatment suggested.

In my hands monoethanolamine nicotinate (nicamin) has been found the simplest, easiest and least time-consuming vasodilator to use from the point of view of both patient and physician. This drug is given subcutaneously, the initial dose being 25 mg. If this dose is well tolerated, the quantity is increased by an equal amount at the daily or twice daily injection until the optimal dose, usually 100 mg. a day, is reached. The patient is instructed in the technic of self-medication and told to continue treatment at home. The injections are continued until the maximal possible improvement in hearing is obtained. The dose is then decreased by increasing the interval between injections. Injections are given every other day for two weeks, three times a week for one week, twice a week for a week and then stopped. If at any subsequent time symptoms begin to return, treatment is again started at 25 mg. and built up to 100 mg. and continued there for one week. Such booster courses are required by the average patient two or three times a year.

For some patients it is possible to substitute nicotinic acid tablets for the nicamin. Fifty to one hundred milligrams once or more daily can be given. Clinical improvement in some patients, however, does not continue when oral administration is used. Beta-pyridilcarbinol (roniacol) given by mouth also has proved to be an effective vasodilator in many instances.

Diet. Kern[20] stated that changes in the water balance of the body may influence the occurrence of allergic phenomena. Increased intake of sodium by tending to retain interstitial fluid, a sort of sodium brine being formed, also will favor the development of allergic reactions. Increased intake of potassium

or decreased intake of sodium by tending to increase intracellular fluid and decrease interstitial fluid on the other hand will antagonize allergic reactions. Beckman[6] considered allergy as a special type of alkalosis. He defined potential alkalosis as the tendency of an individual to store up too much alkali reserve without necessarily having clinical alkalosis. He considered that potential alkalosis is an important causative factor in allergy. As one method of treatment he suggested that patients with an allergy eat fully of fats, meats and cereals, while green vegetables and fruits should be forbidden.

Schemm[32] proposed an ambulatory diet which was not merely a low salt or an acid or neutral ash diet but a combination of all. This diet, modified somewhat, follows:

Maximal intake in twenty-four hours			Minimal intake in twenty-four hours	
Food	Amount		Food	Amount
Milk	1 pint		Eggs	2
Vegetables	2 servings		Meat, fowl and fish	2 servings
			Cereal Foods	Equivalent to 6 slices of bread
Fruit	2 servings except for	→	Prunes, Plums, Cranberries	As desired

1. Avoid adding table salt to foods and avoid foods prepared with salt as pickled herring or sauerkraut.

2. Avoid soda, such as baking soda, soda biscuits, and be careful of artificially softened water and "alkali" water.

3. Avoid salty broths and soups.

I have found that patients can be persuaded to continue to use this diet with relative ease. Restriction of total intake of fluid to about 3,000 cc. daily has been found advisable, together with the use of a diuretic to increase elimination of fluid and sodium. Ammonium chloride in divided doses of 6 to 9 gm. daily has the additional advantage of producing a relative acidity of the tissue fluids.

Banthine. Since allergy appears to be associated with cholinergic preponderance, the use of a drug inhibiting the cholinergic fibers of the autonomic nervous system seems indicated in Ménière's disease. Banthine (beta-diethylaminoethyl xanthene-9-carboxylate methobromide) acts as a blocking agent in the ganglia of the autonomic system. It also has an atropine-like action on the nerve endings of the cholinergic fibers. For this reason its action in therapeutic doses is largely on the cholinergic nerves.

As a full therapeutic dose, 100 mg. of banthine every six hours or 50 mg. of banthine every four hours (night and day) is recommended. After symptomatic relief has been obtained, the dose of banthine may be reduced to 50 mg. every six hours as a maintenance dose. Since stress appears to be an important factor as a precipitating stimulus in Ménière's disease, it is important that the patient be advised to increase his dosage from the maintenance level to the therapeutic level for the duration of any emotional or other stress.

In my hands banthine has been most effective in improving hearing in cases of Ménière's disease with endolymphatic hydrops.

Duration. It is felt that the best results are obtained in endolymphatic hydrops by a combination of these three types of therapy. This combination consists in the elimination of sodium with

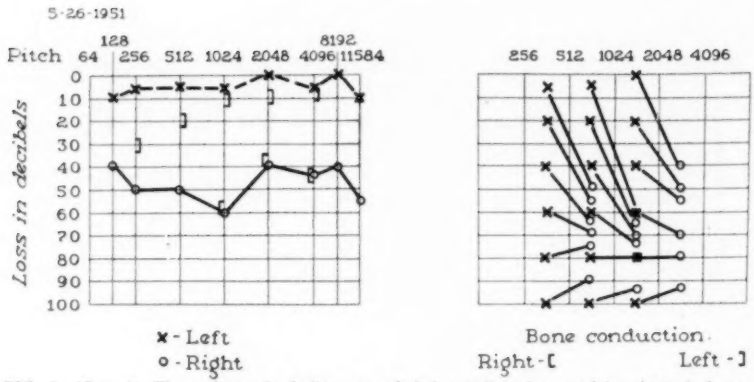

FIG. 3.—(Case 8) The audiometric findings on admission indicated an endolymphatic hydrops in the right ear although the discrimination deafness (fig. 4) was not as great as is often found.

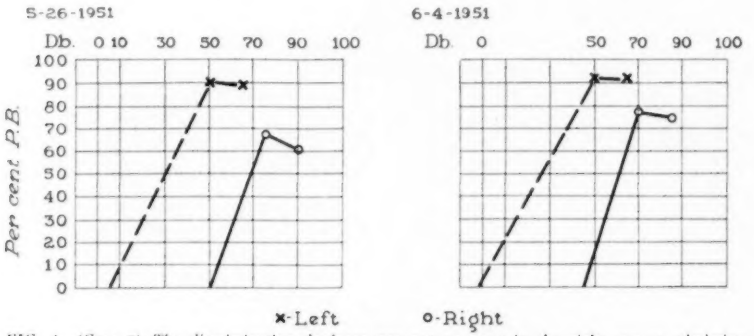

FIG. 4.—(Case 8) The discrimination deafness was not as great in the right ear on admission (May 26, 1951) as it often is in endolymphatic hydrops. By June 4, 1951, there was slight improvement in the discrimination deafness.

an attempt to produce a relative acidosis, the use of a vasodilator to "unlock" the arteriolar spasm and sweep toxic metabolites into the general circulation, and banthine to depress the overactive cholinergic nerves. It is important not to expect too rapid improvement in the cochlear symptoms produced by the hydrops. In a few instances definite improvement has not occurred for four to six weeks after this regimen was started. That improvement may and does eventually occur with treatment is evidenced in the following case.

Case 8. A man, aged 42 years, stated that a year before admission to the clinic his right ear began to buzz. Three months later a severe attack of rotatory vertigo, nausea and vomiting occurred and lasted two hours. Six months before admission he had a second similar attack which was followed by several attacks each week except for a week of freedom two months before admission. He had been continuously dizzy for as long as three weeks at a time.

The hearing in the right ear had been variable during this entire period but on the whole had grown gradually worse. He had found that violent purgation would give considerable subjective relief to symptoms, particularly the deafness. He had many inflations of his eustachian tubes without relief. He had taken dramamine, 50 mg. three times a day, for several months. This had prevented vomiting but had not affected his disorder otherwise.

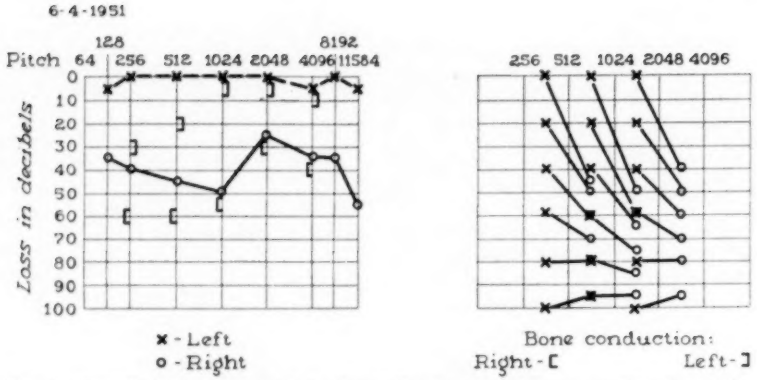

FIG. 5—(Case 8) Little improvement is shown after ten days of treatment although a slight improvement in the discrimination deafness may be noted (fig. 4).

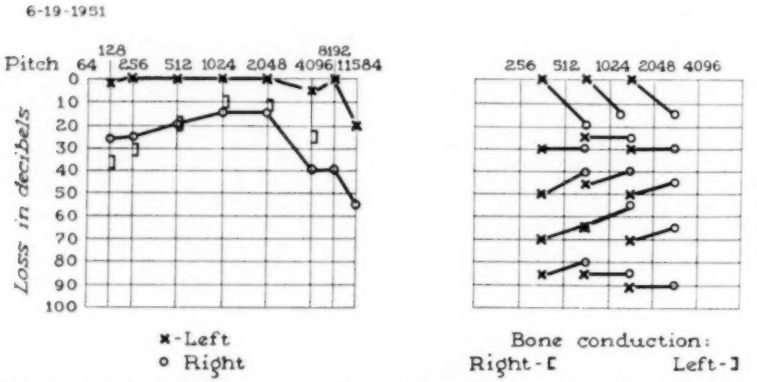

FIG. 6—(Case 8) Definite audiometric evidence of improvement is shown after about three weeks of treatment.

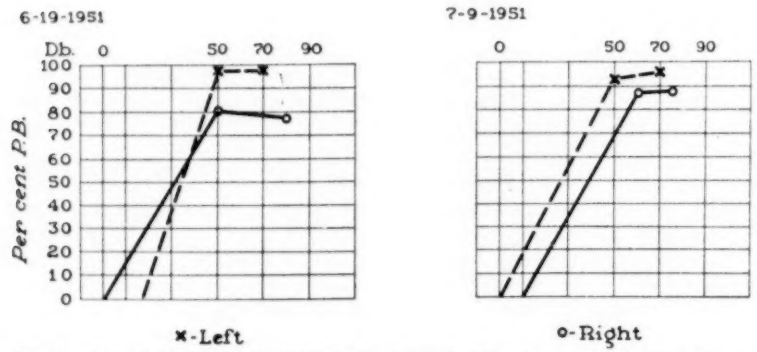

FIG. 7—(Case 8) Discrimination deafness had improved after three weeks of treatment, and more after five weeks of treatment.

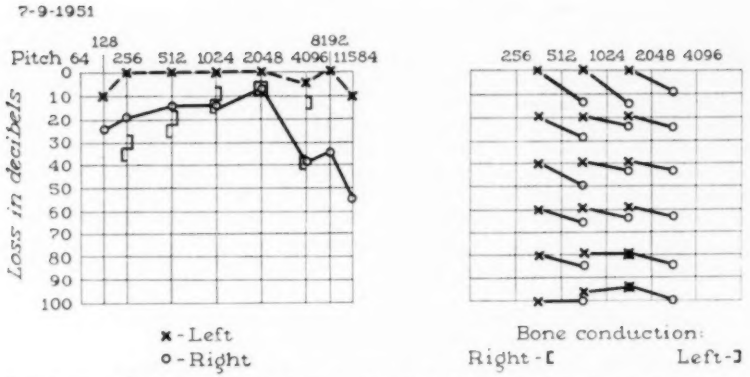

FIG. 8—(Case 8) The audiometric findings after about five weeks of treatment showing slow but steady improvement.

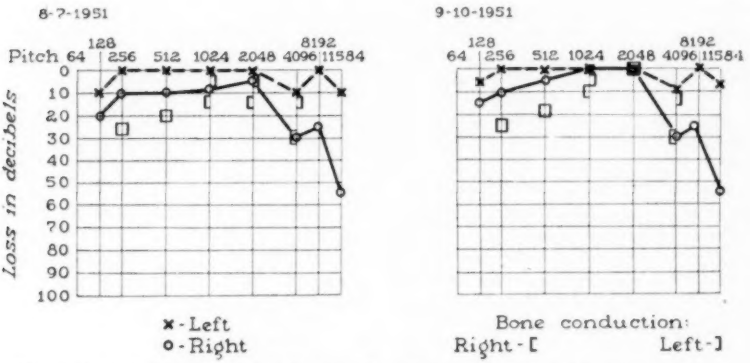

FIG. 9—(Case 8) The threshold audiograms for pure tones by air and bone conduction are shown after three and after four months of continuous treatment.

Ear, nose and throat examination gave negative results except for the findings in regard to the hearing and labyrinthine function. Hearing in the left ear for pure tones at threshold was essentially normal. Hearing in decibels in the right ear for pure tones of various frequencies at threshold showed perception deafness (figs. 3 and 4).

Loudness balance tests showed hyperrecruitment in the right ear over the left at frequencies of 512, 1,024 and 2,048. Testing the function of the equilibrial labyrinth by a modification of the Kobrak test showed marked hypoactivity in the right labyrinth.

This patient received a modification of the Schemm low sodium acid to neutral ash diet, 9 gm. of ammonium chloride daily in divided doses, 50 mg. of banthine every four hours night and day, and 100 mg. of monoethanolamine nicotinate by subcutaneous injection daily. On this regimen he showed no objective improvement in the hearing for eleven days and only a slight improvement in his subjective symptoms. The audiometric findings on the tenth day are shown in figures 4 and 5.

Loudness balance tests still showed hyperrecruitment in the right ear over the left. Improvement, although slight, was definite. The patient was advised to continue treatment at home and to return for observation in two weeks.

On his return he stated that he had been able to work during the previous two weeks for the first time in seven months and that his attacks were infrequent and more like sick

headaches than crises of Ménière's disease (figs. 6 and 7).

Loudness balance tests showed complete recruitment of the right ear at only 30 db. below threshold of the left ear and hyperrecruitment at 50 db.

Speech reception tests done with recordings from the Central Institute for the Deaf showed an improvement in recognition of phonetically balanced words from 65 to 75 per cent. The patient was again sent home to continue treatment for another two weeks. On his return audiometric findings showed improvement (figs. 7 and 8).

Speech reception tests showed a discrimination of 75 per cent for phonetically balanced words in the right ear. The patient was again sent home with instructions to continue the treatment. Figure 9 shows the audiometric findings one and two months later.

The patient had been completely free of any symptoms of vertigo, unsteadiness, dizziness, tinnitus or deafness for the past two months. He was advised to continue with his diet but to decrease the dose of banthine to 50 mg. every six hours and to discontinue the use of ammonium chloride and monoethanolamine nicotinate. He was asked to return for a recheck of his hearing in a month.

This patient illustrates the rather slow progress that can be obtained even with vigorous therapy but shows that if treatment is persisted in, successful results can often be obtained.

SUMMARY AND CONCLUSIONS

The physiologic basis of allergy of the inner ear, usually termed Ménière's disease, is considered in its several variations. These are autonomic dysfunction with its stereotyped or invariable reaction on the peripheral vascular bed, resulting in transudation of an excess fluid high in protein content and consequent elevated osmotic pressure. The vestibular symptoms seem to be mainly due to vasospasm while the cochlear symptoms appear to depend in the main on an increase in endolymph of high osmotic pressure.

The importance of, and some of the pitfalls in, differential diagnosis are stressed and some illustrative case reports are reviewed. Treatment which has given the most satisfactory results at the Mayo Clinic has been a modification of Schemm's diet and other measures to help in the elimination of sodium, administration of a vasodilator to decrease arteriolar spasm and of an inhibitor to depress overactive cholinergic nerves.

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ALLERGY OF THE EXTERNAL EAR

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ALLERGY of the external ear appears in the form of dermatitis, usually of the contact or atopic type. The auricle is more commonly involved than the canal. Pathogenic fungi may also produce dermatitis.

The Auricle

Dermatitis involving the external canal, the auricle and the postauricular area is not uncommon and presents an important problem in diagnosis and treatment. Contact dermatitis is more common than eczema and may be induced by direct contact or by transfer to the affected area by the fingers in the act of scratching the itching area. The contact substance can usually be determined on the basis of the history and patch tests to the suspected substance. It is important to note that a patch test on the forearm may be negative, while if applied to the region of the affected area it may be positive. This is especially true of the skin around the eyes and on the auricles. Here the skin is thin, vascular and more reactive than that on the arms or other parts of the body.

Nail polish dermatitis of the skin around the eyes and the ears is perhaps the most common type. Other cosmetics, such as wave set lotions, may produce similar lesions. The dermatitis may begin as a very small area, which gradually becomes more extensive from scratching and transfer of the polish to other areas. The nails and surrounding skin are usually not affected in these cases. The involvement may be unilateror bilateral. In one instance of nail polish dermatitis, the skin of the ocular regions was involved mostly on the left side. The auricle and postauricular area were involved only on the left side. The greater involvement on the left side was accounted for by the fact that the patient was left-handed.

A great variety of agents, especially those containing dyes and oils, may come into direct contact with the affected area or may be transferred by the fingers. Occupation may be an important factor in these instances.

Direct contact is not uncommon from so-called silver or white-gold spectacles which contain nickel. Plastic frames also may cause dermatitis. Contact with feather pillows may cause eczema on or behind the ear. Secondary infection with pyogenic or fungus organisms may complicate the picture.

The Auditory Canal

Sensitization to medicaments used by topical application to the auditory canal in the treatment of infections not uncommonly occurs. Such preparations as oxide of mercury, merthiolate (other mercurials), sulfonamide compounds, penicillin, streptomycin and similar agents may be the original cause of the dermatitis. In these cases the dermatitis begins in the canal and may spread to the auricle, the cheek or other parts of the body.

The scaling itchy lesions of the auditory canal are usually caused by the fungi of the aspergillus type and are not commonly of allergic origin, although true allergic sensitivity to molds must be considered. Allergic dermatitis, however, may appear from the development

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of sensitivity to some preparation used in treatment.

Mitchell[1] has emphasized the importance of the recognition of low-grade skin infection from hemolytic or nonhemolytic streptococci or from staphylococci. It may be difficult to determine whether the dermatitis is one of primary infection, of allergic origin, or the latter complicated by secondary infection. Patch testing, microscopic examination of scales and exudation, and cultures should be employed in establishing the diagnosis. This applies to lesions of the canal as well as those of the auricle and postauricular area.

Dermatitis from Medicaments

Allergic dermatitis of the auditory canal from some therapeutic agent such as mercurial ear drops quickly spreads to the auricle and surrounding area. Secondary infection, especially in the canal, soon develops. This type of dermatitis may be exemplified by the case of a woman, aged 34 years, who reported with an extensive dermatitis of the entire auricle and surrounding area as well as an involvement of the auditory canal with profuse purulent drainage. The patient stated that about six months previously she had had an acute otitis media for which some ear drops were prescribed for instillation into the auditory canal. After about two weeks the infection appeared to be subsiding but the patient faithfully continued use of the ear drops. Soon thereafter the canal began to itch and a spreading dermatitis of the auricle and surrounding area developed. Upon examination, it was noted that there was no perforation of the drum so the purulent discharge in the canal was the result of an infection of the external canal. Since the ear drops were suspected as the cause of the dermatitis, a patch test was performed on the forearm and it proved to be strongly positive. The ear drops were a proprietary preparation containing mercury. Upon discontinuation of the drops and local treatment of the secondary infection, the condition promptly subsided.

In another case, a man, aged 55 years, was observed for the treatment of a subacute otitis media with questionable mastoiditis. At the onset he was treated by his family physician and the patient did not know the nature of the medication he was given except that it consisted of tablets taken every four hours. This was suggestive of sulfonamide therapy. The patient had no apparent reaction to the drug. After a thorough cleansing of the ear canal it was noted that considerable skin irritation and infection still existed. There was a perforation of the drum with a discharge of mucopus. For local treatment, a preparation of glycerine and sulfathiazole was applied to the canal. The following morning the patient returned, exhibiting marked edematous swelling of the canal and entire auricle with some vesiculation. It was apparent that the patient had developed a contact dermatitis from sensitivity to the sulfonamides. A patch test performed at a later day was positive. Incidentally, the diagnosis was so promptly made in this case that an immediate patch test was not necessary. A large patch reaction might have incited a generalized dermatitis. If the patch test on the forearm were negative in a case of this type, the test should be applied to an area of normal skin on the posterior surface of the auricle. The same applies to cases of dermatitis of the skin of the periocular areas.

The Postauricular Area

Allergic dermatitis may involve only the auricle and postauricular area, thus excluding the canal. These lesions usually commence with vesicular blebs which weep and finally crust. The postauricular involvement usually begins with a fissure in the retro-auricular fold, for the streptococcus has a predilection for

folds in the skin on this as well as other areas of the body. The area constantly weeps with marked crust formation. The scalp also may be affected. The condition may be associated with a seborrheic dermatitis. These lesions are primarily infectious and are usually caused by the streptococcus. It may have spread to this area from an original infection in the external canal or middle ear. Both of these areas may be affected or the original infection in the canal may have subsided. Contact dermatitis or atopic dermatitis is not uncommon. Dermatitis of this area may also be caused by the pathogenic fungi, trichophyton, monilia or epidermophyton. There may be a direct involvement with these fungi or the lesions may be "ids," that is, they are transferred from a primary focus, usually the feet.

Laboratory Diagnosis

Fungi from the affected areas may be identified by cultures or by microscopic examination of the scales or discharge. The scales should be suspended in sodium or potassium hydroxide solution. Streptococci may be recognized by placing the serum or discharge on a slide, fixing with heat and staining with methylene blue or Hansel's stain. The organisms may be identified in the squamous cells in properly prepared specimens. Cultures for streptococci may be made on blood agar plates. Since staphylococci may outgrow streptococci, Vaughan has suggested the addition of 0.06 per cent thallium nitrate or 1 to 100,000 crystal violet to inhibit the staphylococcus, thus permitting the streptococcus to grow more abundantly. Crystal violet decomposes on autoclaving, and should not be added until the time of inoculation. Mitchell[1] recommends the use of brain broth glucose medium in tubes at least 12 inches in length. Crystal violet may be added. Subcultures should be made after about 18 hours.

Treatment

The treatment of the various types of dermatitis of the external ear depends upon the cause of the trouble and the management of complications. Primarily the allergic dermatitis is an atopic eczema, which may be observed in infants and children, or contact dermatitis. In addition to the use of local treatment, the management of eczema is the same as that which appears on other parts of the body. Secondary fungus or pyogenic infections must be recognized when present and proper therapy directed to their elimination.

In the cases of contact dermatitis it may be possible to avoid or eliminate the causative agent. In the case of cosmetics or drugs used in local treatment, the substitution of some other subtance may be possible. The prolonged application of a new substance to the skin may result in the development of a sensitivity to it.

When the etiologic agent is a plant oil such as poison ivy or some type of flower or weed, the oil fraction may be administered orally or sublingually or by injection for immunization. Any substance which is not poisonous can be used in therapy in the same manner.

Immunization therapy. Oral immunization for plant dermatitis has been used extensively by Shelmire.[2] The method consists in starting with a small dose and gradually increasing to the point of relief or tolerance. Small dose sublingual or oral therapy with dilutions of the causative oil fraction in sesame oil has been most satisfactory. Optimum doses range from the 1:10,000 to the 1:10,000,000 dilutions, with the 100,000 and 1,000,000 as the average. (One drop every one or two days.)

Staphylococcus toxoid in intracutaneous doses of 0.1 to 1.0 unit at three to four day intervals has been very effective as a nonspecific agent in the treatment of eczema or contact dermatitis.

For complicating staphylococcus infection, these same small doses are effective in giving immunity. Streptococcus infections could be treated in the same manner.

Fungus infections may be eliminated by local treatment, or injection of fungus extracts may be necessary to produce immunity. The oral administration of iodides may be of considerable benefit. In some instances, actual fungus sensitization, rather than infection, may be the answer to the problem at hand. In either event, the injection of fungus extracts at five to seven day intervals may be advisable.

We have employed a mixture of equal parts of trichophytin, oidiomycin and epidermophytin in dilutions from 1:100,000 to 1:10,000,000,000 in the treatment of these cases with most satisfactory results. A combination of this fungus mixture and staphylococcus toxoid may be employed.

In instances of marked complicating pyogenic infection, the sulfonamides or antibiotics, such as penicillin, may be employed. Local application of these drugs in the form of ointments may be all that is necessary. Thorough cleansing of the lesions should always precede the local application. Prolonged use of these or any other preparations over a long period of time may result in contact dermatitis from them. Various types of ointments such as Whitfield, pragmatar, salcresin or tar ointments to relieve itching may be a necessary part of the treatment, but here also sensitivity may develop to the substance applied.

The development of sensitivities of this type may be masked or unrecognized and at some later time an acute local or general exacerbation of the dermatitis may be excited when the substance is readministered orally or by injection. This occurrence has been particularly noted in the case of the use of penicillin and the sulfonamides. These points should receive due consideration in the postoperative management of mastoid cases.

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AURAL ALLERGY

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ALLERGY is a word that was coined to designate an altered state of an organism so that its victims respond in a most unusual manner when it comes in contact with certain materials in amounts that otherwise are not harmful. Consequently an allergy must be distinguished from an idiosyncrasy or an intolerance. In these two, the response is the usual one but the amount of exposure (dose) that is necessary to produce the full physiologic effect may be very much less than the usual one. For example, every human being will get a ringing in his ears if he is given enough quinine. This is, then, a normal response to quinine, as it is to streptomycin, no matter how much or how little it takes to produce it. This we may term an idiosyncrasy or intolerance. A small percentage of individuals, however, when given quinine break out with hives. Now, hives is not the usual response to quinine. Something has altered the state of these persons and so we use another word for it and call it allergy to quinine.

We recognize, then, that in allergy we are dealing with a condition or state of the person which may result in disease when the victim comes in contact with the substance to which he is now allergic — his allergen. Modern medicine has learned to control these allergic reactions and thus do away with them through the use of avoidance, elimination, substitution, identification programs, or by overcoming the allergic condition through the use of graduated injections — hyposensitization, as it is called. In these ways allergic persons are kept comfortably relieved from their debilitating exacerbations. The medical profession knows much less about changing the allergic constitution back to normal. There is hope today that the newer drugs, ACTH and cortisone, may become the tools by which this problem will be solved, but all that is for the future. At the present time they present an opportunity for protection against the ravaging symptoms of acute episodes but offer no lasting improvement.

The allergies of the nose and ear are unusual reactions of the nose and ear tissue to an allergic stimulant which is different from the ordinary reaction that one would expect from the same or a similar stimulus. These are manifested in peculiar ways and interfere with the normal function. The symptoms of nasal allergy are itching, sneezing, obstruction from swelling of the mucous membrane, watery discharge, pain from the blocking of the ostium of the sinuses, and the promotion of secondary infections in the sinuses or the development of nasal polypi if the stimulant continues. One can say that nasal polypi are always due to allergic reactions of nasal mucous mmebranes. If any inhalation or injection of a specific allergen causes a dullness, fullness, pain in the ears, lowered hearing, tinnitus, increase or change in the discharge in an otorrhea, allergy of the middle ear must be considered.

Diagnosis: The history of inheritance of allergies varies in its manifestations in different members of the same family, but about 10 per cent of the population has allergies of one type or another.

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The history of the disease and its attacks includes in the nasal pathologic findings a preponderance of eosinophils in the nasal secretion. The nasal polypi that are removed from allergic noses are found on section to contain many eosinophils, also, in addition to their watery mucoid structure.

Treatment: In the management of an established nasal sinusitis and polyposis it does not follow that the treatment of the allergies will always completely correct the situation. One must be certain that the proper therapy for each particular case is instituted. This consists of reducing the infection by antral lavage, general physical build-up, decongestant applications to the nose, suction for removal of excessive secretions and careful allergic studies to determine the cause of the disturbances. The primary disturbance is usually of allergic origin and the secondary disturbance of infectious origin. From the surgical standpoint, it is important that the normal anatomic status of the nose be reestablished in addition to the corrective medical measures of returning nasal physiology to normal. If the nose is filled with polypi, they need to be removed as an initial step in the procedure. If the antrums and ethmoidal sinuses are loaded with polypi and the normal mucous membranes are nonfunctioning or are destroyed, it is necessary to remove them. The management of infections in allergic patients does not vary from the management of nonallergic infections except that additive measures are employed. In cases of deviated septums with nasal obstruction and difficulty in breathing, there is a great relief obtained by a submucous resection of the nasal septum. In fact this offers one of the greatest helps in the commonest nasal obstructions.

Allergic disease of the ear should be divided into three groups: allergies of (1) the external auditory canal and adjacent tissues, (2) the middle ear, and (3) the inner ear.

THE EXTERNAL EAR

Allergic otitis externa is a dermatitis characterized by hyperemia, inflammatory reactions, edema, and in severe acute cases, vesiculation. In the more chronic types, eczematoid changes occur. Allergic dermatitis is more frequent than is generally supposed and is due to the effect of some contact irritant or allergen to which the patient is specifically hypersensitive.

Fur neckpieces or fur collar trimmings are fairly frequent causes for reaction about the ear. Cosmetics are suspect, particularly those containing orris root, i.e., face powder, face cream, lotions, and the like. Intermittent exposure to an irritant may give rise to a puzzling otitis externa which varies in severity from day to day. Fingernail polish, scalp lotions, medicaments used about the ear, perfumes, essential oils, phenol, cresylic acid, the temple pieces of spectacles, and sulfonamides may be offenders. Treatment resolves itself into the elimination of the irritant responsible for the lesion if it can be discovered, correction of deficiencies in diet and vitamin intake, and the use of such local applications as are necessary to aid in correcting the changes which have taken place in the skin.

Care should be exercised in the choice of local applications and treatment, for the drug that is intended to clear up a local situation may through its irritant properties serve to perpetuate it. The diagnosis of external otitis frequently, even if of an allergic nature, is considered to be due to fungus growth. However, though this does occur rarely as a primary cause, it usually occurs rather as a secondary invasion, as does involvement by many saprophytic bacteria. The list of local applications for the correction and palliation of the itching, burning, discharge and dermatitis that fol-

low, and which are usually secondary, is a long one in the history of medical treatments, ranging from hydrogen peroxide and many combinations with it, through the sulfonamides to zinc oxide and on through nearly forty remedies. However, very careful cleanliness and repeated treating of the entire external ear with dry applicators or with applications of pure alcohol will quiet down the local manifestation and control the situation until the allergic factors can be taken into consideration and relieved by eliminating the irritants responsible for the situation, if and when they are of an allergic nature. We use small doses of staphylococcus toxoid, 0.1 to 1.0 unit, and when fungi are discovered, small doses of 0.1 to 0.5 of a mixture of tricophytin, epidermophytin and monilin in a dilution of 1:100,000. Frequent applications of silver nitrate, 2 to 20 per cent, to the abraded areas is also helpful. A neurodermatitis and a seborrheic dermatitis are frequently noted here. They appear much the same and can be differentiated by giving doses of cortisone. The neurodermatitis disappears in two or three days and then promptly recurs when the cortisone is stopped. The seborrheic dermatitis is best controlled by a 2 per cent sulfur ointment.

THE MIDDLE EAR

Allergies of the middle ear frequently cause change in the hearing. This is frequently of the combined lesion deafness type, i.e., involving both high and low sound frequencies, but oftener it is in the high frequencies only, thus simulating a nerve deafness. The loss of hearing is dependent on the location of the affected or shock tissue, and the commonest symptoms are deafness, fullness, dullness, deep-seated pain, itching between the nose and ear and a burning sensation in the ear. In our experience, excluding the senile deafness cases, we find a distinct diagnosis of allergic deafness in approximately 2 per cent of hearing difficulty cases. They are found for the most part in the first twenty years of life. The diagnosis is rather easily established by making, first, a careful audiometric recording. The patient is then given a hypodermic dose of adrenalin, and the audiometric test is repeated in fifteen to twenty minutes. If there is an immediate change in the hearing, one can infer that there is probably an allergic factor and be on guard to determine the allergens responsible. Allergic therapy can then be added to the other therapies that are indicated. We have found the commonest allergens to be inhalants and foods.

Catarrhal Otitis Media

In the deafness that comes with a secretory catarrh of the middle ear it has been noted for a long time that the secretion frequently contains eosinophils. These episodes are often accompanied by an acute nasal allergy. The relief of this type of situation is much quicker and the repetition of attack much less likely if the allergic factors involved can be sought out and avoided. A repeated catarrhal otitis causes insidious permanent hearing changes.

Chronic Recurrent Catarrhal Otitis

In a recurrent catarrhal otitis with its resultant hearing losses, careful cytology studies of the middle ear fluid are essential and often add greatly to the efficiency of therapy. I remember seeing a young man, 20 years old, some years ago who had had 21 myringotomies in five years, for 21 different attacks of catarrhal otitis. His drums were thin, scarred, and had some calcereous deposits, giving them a ground-glass appearance. Yet he had very little loss of hearing. No smears had been made on the fluid from these myringotomies and no smears of his nasal secretions had been made either. He was cured of these yearly recurrences by having his nasal polypi removed, dust desensitization,

and by avoidance of tobacco. Recently a confrere from a distant city reported that he had seen this same patient five years later, after a climate change, for some asthma and nasal allergies which again cleared up by proper allergic therapy without recurrence of his middle ear involvement.

Radium application. A very fine result in some hearing difficulties in children has been accomplished by the application of radium to the nasopharynx to cut down the hypertrophic lymphoid tissue, thereby destroying its possibility of encroachment on the eustachian tubes, thus obstructing the middle ear. I believe that those cases which get a fine therapeutic result from radium are often allergies and that the use of radium has for its effect the nonspecific reaction of traumatized tissue in addition to the reduction of the lymphoid tissue. In many instances where we have obtained good results we have been able to find a high eosinophilia in the nasopharyngeal secretion.

Eosinophilic and Aneosinophilic Ear

In chronic otorrhea if the perforation is central, eosinophils may be frequently found in the secretions, and unless the etiologic factors are discovered and removed by avoidance or hyposensitization, these will remain chronic and undergo frequent episodes with changes in the secretion with every acute allergic episode and cold. They will show very slow healing if the mastoid itself is invaded, and surgery becomes necessary.

The aneosinophilic ear usually has a marginal perforation and bone changes with necrosis or cholesteatoma. When carefully cleaned out, these heal much quicker than the eosinophilic ears.

When middle ear tissues are sensitized locally early in life they have a developmental inhibition to good pneumatization and normal development of the middle ear and mastoid air cell system and increase thereby the opportunity for chronic otitis and mastoiditis.

It has been considered by Dohlman[2] that when babies are artifically fed they lie flat on their backs, and as they have straight eustachian tubes this offers one of the best opportunities for local sensitization to cow's milk. In his series of cases, 86 per cent were artifically fed and indeed the commonest sensitivity was to cow's milk. So we see that the middle ear can be assaulted from an allergic standpoint either by foods or inhalants or by local sensitization.

INNER EAR

Allergies affecting the inner ear have the following symptoms: tinnitus aurium, vertigo, unpleasant clicking noises, and unilateral or bilateral hearing defects.

Endolymphatic hydrops is the syndrome of physical or intrinsic allergies. In a discussion of allergies of the cochlea and labyrinth, commonly referred to as Ménière's syndrome, there is a great difficulty in sharply dividing and classifying the symptoms. There may be vertigo, nausea and vomiting, with or without hearing loss. The typical Ménière's diagnostic triad is vertigo, tinnitus, and deafness. There is much evidence to justify the classification of a Ménière's syndrome as an allergy, and it frequently involves other structures of the head with accompanying myalgias and neuralgias. There are some cases of pathologic sections showing dilatation of the endolymphatic system, degenerative changes being present in the sensory elements of the inner ear and showing no inflammatory changes. Pathologic specimens and descriptions are reported by Hallpike and Cairns in England and John Lindsay of Chicago.

A characteristic "endolymphatic hydrops" is a noninflammatory extracellular edema fluctuation and is quite consistent with an allergic etiology. It is difficult to find the pathologic picture

of a fluctuating extracellular edema in the body without having an allergic background or etiology. The disease usually starts in middle life, sometimes as early as the third decade. Vertigo may be the first symptom, but often it is tinnitus and a deafness that is frequently profound and quick in its onset, the loss of hearing amounting to as much as 40 decibels in a few minutes. The deafness may involve only the lower frequencies, but it is usually progressive and actually involves all the frequencies for both air and bone conduction. It may affect one or both ears, and it occasionally happens that there may be little or no vertigo. The tinnitus and deafness progress in a typical manner. Treatment consists of careful allergic study, elimination diet, histamine diphosphate injection and rest. In uncontrolled cases the destruction of the labyrinth must be accomplished.

CONCLUSIONS

Nasal allergies are quite common in the practice of ear, nose and throat.

Allergy of the middle ear is an unusual reaction to an allergic stimulus and is characterized by changes in hearing, catarrhal otitis, and eosinophilic otorrhea.

Surgical intervention is sometimes avoided and its efficiency many times increased in both ear and nasal conditions by recognizing the allergic basis of the pathologic conditions.

A test is given for the diagnosis of allergic deafness.

Discussion of inner ear deafness and indications of treatment are suggested.

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THE ALLERGIC FACTOR IN CHRONIC BRONCHOPULMONARY INFECTIONS

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THE behavior of the tissues of allergic individuals in response to infection or other form of stress is different from the response of the tissues of nonallergic people to similar insult.

Kline[17] has pointed out that the characteristics of infections in allergic people are rapid onset, violent course, and slow regression. Kline says the tissue changes in allergy may be considered under five headings: (1) functional changes, (2) inflammatory changes, (3) proliferative changes, (4) degenerative changes, and (5) necrotic changes. It is my belief that the necrotizing type of allergic reaction described by Kline is responsible for the development of many cases of bronchiectasis, bacterial proteins being the antigen responsible for the reaction. Upon reviewing the charts of 50 bronchiectatic patients,[6] I found that 80 per cent of them were allergic individuals. This corresponds closely with the figure given by Watson and Kibler,[24] who found that 82.6 per cent of their bronchiectatic patients had allergic manifestations. The implication is not that sensitivity to foods or inhalants have much, if anything, to do with the development of bronchiectasis, but that persons with an allergic constitution are easily sensitized to bacterial proteins and thus apt to develop the necrotic type of allergic reaction described by Kline.

Kahn indicates that allergy is an expression of hyperimmunity and implies that it is an overaction of a physiologic mechanism. Kahn[16] defines an allergic individual as one who develops immunity more readily, and to smaller quantities of an antigen, than does a normal individual. I agree with H. L. Williams' statement[25] that since the publication of "Tissue Immunity,"[16] no evidence has been produced to indicate any inaccuracy in Kahn's conclusions.

One of the apparent benefits deriving from the use of cortisone in these allergic individuals is its capacity to reduce the physiologic over-reaction to infection. More specifically, its use decreases the excessive mucosal edema and submucosal infiltration with leukocytes, thus widening the bronchial lumen, relieving the dyspnea, and permitting better drainage.

An instance of hyperimmunity or over-reaction to infection or other irritant may have no serious consequences if it takes place on a cutaneous surface, but when it takes place within the confines of a bronchial tube the resulting obstruction has serious consequences, causing dyspnea, bronchial obstruction, and often atelectasis, which if long continued can lead to bronchiectasis. This statement implies the need for early and adequate treatment by measures to be described, including bronchoscopic aspiration.

These remarks are made from the point of view of a bronchoscopist who perhaps has only a small knowledge of allergy, but who recognizes the importance of attempting to evaluate allergic factors in the diagnosis and treatment of chronic bronchopulmonary infections.

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H. L. Williams[26] describes three related, but not identical types of allergy: (1) physical allergy, (2) bacterial or tissue allergy, and (3) humoral allergy. In my opinion, the chief difficulty of these individuals with chronic bronchopulmonary infection is the tuberculin type of bacterial allergy described by Swineford and Holman[21] and by Scherago.[18] In addition to the bronchopulmonary infection these patients frequently have foci of infection around the teeth, in the tonsils or sinuses. Adequate management of these patients with bacterial allergy includes elimination of *all* foci of infection. Effective treatment usually requires surgery plus bactericidal doses of penicillin. Grorud[12] says the antibiotic therapy of intrinsic asthma is not as valuable as was first anticipated. I think this is true because few men have used bactericidal doses. Bronfenbrenner[2] says the basic mechanism involved in bacterial allergy is identical with that of allergy to simple proteins. My remarks apply only to nontuberculous pulmonary disease.

In an attempt to elucidate etiologic factors, I studied the charts of 25 asthmatic patients for whom bronchoscopy was recommended either for diagnosis or for treatment. The age incidence is informative, 1 patient being in the first decade, 1 in the second, 2 in the third, 1 in the fourth, 6 in the fifth, 10 in the sixth, 4 in the seventh. Thus 20 of the 25 patients were over 40 years of age. This is in line with the observations of Brown[3] and Forman[10] that allergic disease that begins after the age of 40 years is almost always due to bacteria. All of the 25 asthmatic patients for whom bronchoscopy was deemed necessary had chronic suppurative bronchitis, which indicates that the infectious factor must be vigorously treated if their symptoms are to be relieved. Four of the 25 in addition had bronchiectasis. In each of these 4 cases there was a clear history of pneumonia or pneumonitis which initiated the bronchiectatic symptoms. This is in line with my previous observation[7] that patients having bronchitis and/or asthma never develop bronchiectasis unless they have had an episode of acute destructive pneumonitis. It also lends weight to the idea that bronchiectasis can be prevented if acute bronchopulmonary infections in allergic individuals are promptly and vigorously treated with *bactericidal* doses of the appropriate antibiotic or combinations of antibiotics. Large doses are necessary because many of the cocci have become relatively resistant to the action of penicillin.[8] Thus the prevention of bronchiectasis is the serious responsibility of the physician who first sees an allergic individual with an acute bronchopulmonary infection. My usual formula for treatment of such patients is intramuscular injection of 800,000 units of fortified procaine penicillin every four hours, omitting the 4:00 a.m. dose, plus 0.5 gram of streptomycin intramuscularly every 12 hours. In vitro studies have shown that mixtures of streptomycin and penicillin have a synergistic action. I have not placed much reliance on aureomycin and terramycin because they are bacteriostatic rather than bactericidal. This probably accounts for the relatively poor clinical results despite the high degree of in vitro sensitivity shown by most of the gram-positive cocci to these two antibiotics. In addition, I think it is helpful to administer cortisone, 25 mg. by mouth, every four hours to decrease the excessive allergic inflammatory reaction which causes bronchial obstruction and retention of exudate. The possible benefit deriving from combined use of cortisone and penicillin was suggested by Selye.[19]

I think it is bad practice to use antibiotics topically because of the danger of sensitization. I had several asthmatic patients who were made distinctly worse

by penicillin aerosol inhalations so I quickly discontinued this type of therapy.

Five of the asthmatic patients had, in addition, Loeffler's syndrome or allergic pulmonary consolidation, of the type described by Hansen-Pruss and Goodman.[14] Thirteen, or approximately half, of these asthmatic patients with bronchitis also had chronic suppurative sinusitis. The frequent coincidence of sinusitis and infectious asthma does not prove that one caused the other. My interpretation is that bacterial allergy is responsible for the chronicity of the infection in both the upper and lower respiratory tracts.

The indications for bronchoscopy in asthmatic patients can be considered under two headings: (1) as a means of diagnosis, and (2) for therapeutic benefit by aspirating the viscid exudate which the patient has been unable to cough out. In regard to diagnosis, bronchoscopy is of value in ruling out other causes of wheezing. It also gives a rather clear idea of the degree of bronchitis present and the degree of mucosal thickening, but more important, it furnishes an uncontaminated specimen of endobronchial exudate for smears and cultures, including sensitivity cultures, which I think are important in indicating the choice of antibiotic to be used and the dose which will probably be necessary to eradicate the infection. In this series of 25 cases, 16 or 64 per cent, had at least temporary therapeutic benefit by removal of the gummy endobronchial exudate. This benefit was especially noticeable in the patients who had Loeffler's syndrome.

Three of the patients were made distinctly worse by bronchoscopic examination because the procedure induced severe acute bronchospasm, and one of these patients died shortly after the bronchoscopic examination. This bronchoscopy was not performed by me. On reviewing the chart it seems evident that bronchoscopy should have been deferred until his extreme state of exhaustion and dehydration had been corrected by medical means.

Three of the patients with Loeffler's syndrome were thought from the radiologic examination to have tuberculosis, but the repeatedly negative sputum examinations and prompt clearing of the pulmonary densities ruled out the latter.

A review of the case histories of several of these asthmatic patients referred for bronchoscopic examination will, I think, illustrate the infectious bronchitis which in my opinion is the chief etiologic factor in those patients who require bronchoscopic examination.

Case 1. I. Y., aged 52, was admitted on our service in February 1950. She said that her asthma began after she had had an attack of "influenza" 20 years ago. Her asthma had been intermittent and sometimes for an entire year she would not have any wheezing or other evidence of asthma. Three months prior to admission she developed bronchitis and marked persistent wheezing with difficulty in expiration. She had a loud expiratory wheeze which suggested some type of bronchial obstruction other than bronchial asthma. There were musical rales throughout both sides of the chest. Bronchoscopic examination showed rather marked diffuse congestion and edema of the tracheobronchial mucosa and the mucosa was considerably thickened, as evidenced by the fact that the spurs were much wider than normal. A moderate amount of mucopurulent exudate was aspirated and sent to the laboratory for smears and cultures. There was no visible obstruction of any of the branch bronchi other than the partial obstruction produced by generalized mucosal edema and submucosal infiltration. Smears of the endobronchial exudate showed many pus cells but only a few eosinophils. Culture showed a hemolytic Staphylococcus aureus which grew on a 3-unit penicillin plate, was inhibited on a 5-unit penicillin plate, and on the 5-microgram streptomycin plate. She was treated with fortified procaine penicillin, 400,000 units every three hours, and streptomycin, 0.7 grams every 12 hours, for a period of seven days. She was totally free from cough and wheezing when she left the hospital at the end of a week's treatment, so it

seems evident that the antibacterial therapy was effective. Bronchoscopy was useful in this case in focusing attention on the bacterial bronchitis and in furnishing an uncontaminated specimen for smears and sensitivity cultures. Seventy skin tests were made by the method described by Walker (23), and all were negative except the reaction to timothy and to several of the June grasses, which were positive. She had no increase in symptoms during the summer months, so it is evident that she did not have clinical sensitivity to pollens. Roentgen examination of her chest showed no abnormality other than the presence of a few small calcium deposits in the region of the right middle lobe. She had no recurrence until she developed bronchitis seven months later.

Case 2. E. S., aged 44 years. This patient had an acute exacerbation of chronic suppurative bronchitis. He had bronchial asthma of approximately ten years' duration and his asthma had been worse for two weeks prior to admission to the hospital. Roentgen examination showed marked bullous emphysema. Bronchoscopic examination showed acute diffuse congestion of the tracheobronchial mucosa and several cubic centimeters of thick mucopus were aspirated. Introduction of the bronchoscope caused considerable increase in his bronchospasm and produced cyanosis. Bronchospasm developed even though he had been given aminophylline intravenously and adrenalin subcutaneously just prior to the bronchoscopy. The events in this case report suggest that bronchoscopic aspiration should be deferred as advised by Hansel (13) until the acute bronchitis is controlled by medical means. This patient was treated with fortified procaine penicillin, 400,000 units every six hours, and streptomycin, 0.7 grams every 12 hours, as well as by aminophylline administered by slow intravenous drip twice daily. His temperature, which had been 101C., came down to normal and he left the hospital much improved at the end of five days.

Case 3. H. C., aged 67. This patient had had recurrent asthma since she was 38 years of age, but it had been almost continuous for six months prior to admission and required adrenalin injections for relief. She had been coughing up one ounce of yellow sputum daily. There were loud wheezes throughout both sides of the chest. Roentgen examination showed considerable emphysema and segmental atelectasis in both lower lobes. Bronchoscopic aspiration yielded several cubic centimeters of extremely thick, gummy, yellow mucopus. There was moderate diffuse congestion and edema of the tracheobronchial mucosa. Smears from the endobronchial exudate showed many gram-positive cocci and many pus cells. No eosinophils were seen. Culture showed a Streptococcus viridans which grew freely on the 1-unit penicillin plate, but which was inhibited by the 3-unit penicillin plate, as well as by the 5-microgram streptomycin plate. She was treated with fortified procaine penicillin, 400,000 units every six hours, and streptomycin, 0.75 grams every 12 hours, for a period of seven days. Her asthmatic symptoms cleared up completely and the patient said her health was the best it had been in the preceding two years. A follow-up report 15 months later indicated that she had had no recurrence of asthma, had not needed a doctor and enjoyed doing all of her own housework.

Case 4. K. Q. This patient, aged 52 years, had asthma which developed immediately following an acute upper respiratory infection two months prior to admission. The asthma has been so bad that she had to sit up in a chair all night. She had a moderate amount of mucopurulent sputum. She never had had any previous asthma or any type of allergic symptoms. She was treated in another hospital with penicillin, epinephrine and oxygen, without relief, so her doctor thought her asthma might be on a cardiac basis and gave her digitalis. There was no family or personal history of allergy. The blood count showed 24 per cent eosinophils. Roentgen examination revealed consolidation in the superior segment of the left lower lobe. Bronchoscopic examination four days later showed congestion and thickening of the bronchial mucosa with the bronchial lumen markedly diminished on expiration. About 2 cubic centimeters of tenacious yellow mucopus were aspirated. The bronchologist made a diagnosis of Loeffler's syndrome and advised treatment with ACTH.

A second roentgen examination, a week after the first, showed 40 per cent clearing in the shadows of infiltration and consolidation in the left lung and a new area of increased density in the right middle lobe. Another roentgenogram at the end of the second week showed normal lungs. She had fever up to 101 C. prior to bronchoscopic aspiration and administration of ACTH. Thereafter her temperature remained strictly normal. In addition, she received aminophylline by slow intravenous drip. No aminophylline was needed after the first week, or two days after bronchoscopic aspiration and beginning of administration of ACTH. A follow-up report three

months later stated that she had not had any recurrence.

A review of the charts of these patients with chronic suppurative bronchitis and asthma is depressing because so many of them have recurrences. Nearly all of them had very gratifying response to treatment with large doses of penicillin and the other measures described over a period of seven to ten days. Most of them would remain free from symptoms for periods varying from one to six months, until another respiratory infection again precipitated asthma. In other words, treatment with penicillin does nothing to prevent the next infection. The same temporary type of benefit is evident in the patients who receive either ACTH or cortisone.

On the title page of the book by Sir Almroth Wright,[27] entitled "Studies on Immunisation, Second Series," is the statement: "The physician of the future will be an immunisator."

I must confess that vaccine therapy is one type of treatment I have neglected in the management of cases of this type since penicillin became available. My recent reading has given me the conviction that it is a type of treatment that can benefit some of these patients. The effectiveness of vaccine therapy has been pointed out by Cooke,[5] Clerf,[4] Wright,[27] Brown,[3] Silcox,[20] Gay,[11] Unger,[22] Zinsser,[28] Eagle,[9] Benson,[1] Heise[15] and many others. I think vaccine therapy should be used when removal of foci of infection and high dose antibiotic therapy have failed to cure the patient.

CONCLUSIONS

Asthma that begins after the age of 40 usually results from infectious bronchitis and is due to the tuberculin type of bacterial allergy. Adequate management of these patients having bacterial allergy includes elimination of *all* foci of infection.

Bactericidal doses of penicillin combined with the usual antiasthmatic measures will frequently clear up the pulmonary infection if other foci of infection are eliminated by surgery.

Bronchoscopic examination is of value for both diagnosis and treatment. It focuses attention on the infectious bronchitis, which is apt to be minimized or overlooked in asthmatic patients.

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DISCUSSION

PAUL H. HOLINGER, M.D., Chicago, Ill.: Dr. Davison's excellent presentation has been a little too modest in that he has failed to stress the important contribution he has made to this subject in previous papers presented before the American Bronchoesophagological Association and other groups, illustrating the importance of the relationship of allergy to bronchiectasis. In these presentations he has pointed out the significant basic role of allergy in the patient who has a sinusitis in association with his bronchiectasis. His statistics show that a high percentage of patients who have the so-called sinopulmonary syndrome have allergy, and that in patients who have either a sinusitis alone or bronchiectasis alone, a relatively low percentage have allergy. I think this becomes an important and significant phase of his present discussion, and possibly he will elaborate a little on this in his closing remarks.

His remarks in regard to allergy as being responsible for bronchiectasis had, in this presentation today, a new etiologic slant; not that allergy has not previously been considered in the etiology of bronchiectasis through edema of the mucous membrane and bronchial obstruction, but in his theory of local destruction. This is an interesting feature, and possibly we can have a little more discussion in regard to the exact evidence of the destructive nature of allergy in the area below the point of obstruction.

Dr. Davison stated that the lesions are found more frequently in patients over forty. Is there any explanation as to why age enters into this problem? In our own experience, children who have allergic changes associated with bronchiectasis are seen just as frequently as adults; and the whole problem described in adults has been duplicated in children. However, sinusitis and bronchiectasis are not seen together so frequently in children. That is, the child whose bronchiectasis began early in life very rarely has sinusitis in association with it. It is only later that the child develops sinusitis; but, as Dr. Davison suggests, only the children who have an allergic background seem to develop the combination of sinusitis and bronchiectasis.

One of the other features of the presentation that interested me was the discussion of the eosinophil count in the patients mentioned. In the first patient, the eosinophil count in the secretions removed bronchoscopically was apparently nil; one wonders, therefore, whether this was simply a suppurative process without any perceptible allergic background, in spite of the fact that the patient had "asthma," and that this was not truly an allergy. I would appreciate it if Dr. Davison would discuss this further.

In our presentation before this group last year, suggested by Dr. Hansel's remarks two or three years previously, we reported the value of determining the percentages of eosinophils and neutrophils in patients with bronchiectasis and other bronchopulmonary suppuration. It was found that the ratio of eosinophils to neutrophils followed the studies that he had made on the secretions in the

nose: the patient who had a suppurative bronchitis of a bacterial character without any allergic background had a very high percentage of neutrophils, whereas the patient who had an obvious allergy had a high percentage of eosinophils in the bronchoscopically obtained secretions. One of the discussions presented this morning showed the same thing in a slightly different manner.

In regard to problems of chemotherapy in chest work, the newer phase of that work concerns the increasing number of patients who are developing fungus and pyocyaneous infections with the use of chemotherapy over a long period time. I was glad to hear Dr. Davison stress the fact that large doses of the chemotherapeutic agents should be used for a relatively short period of time, rather than maintenance of the patient over a long period of time on smaller doses of chemotherapy, while the organisms responsible for the original infection disappear and are substituted by other organisms that are much more difficult to eradicate than the original infection.

The vaccines that Dr. Davison mentioned are significant and important in patients with a true bacterial infection in the tracheobronchial tree, and we feel they are a definite adjunct to treatment.

I was exceedingly interested in this paper because it presents newer phases of a subject which is a very common one, and one which requires the combined efforts of the chest group, the rhinologists, and the allergists as well as the internists in order to get rid of the bronchopulmonary suppuration.

FREDERICK SIMMONS, M.D., Marion, Ind.: Dr. Davison has had very good results in the treatment of his reported cases of suppurative bronchitis, for which he deserves much credit. As to the mechanism causing the disease process and its subsequent recurrence, factors other than bacterial allergy come into play and should be mentioned. According to Dr. Davison's report, the majority of his cases (of suppurative bronchitis resistant to the usual methods of treatment) occur in the fifth decade of life, or later, which corresponds to our experience.

It would seem to me that chronic suppurative bronchitis is due to infectious changes occurring in a patient already showing the degenerative changes accompanying age, leading to a marked mechanical obstruction of the bronchus. To be more specific, practically all aging people develop some degree of alveolar emphysema, and often other changes such as pulmonary fibrosis and chronic bronchitis. Nonobstructive senile emphysema is due to skeletal changes, chiefly in the thoracic spine. Thinning of the intervertebral disks causes shortening of the spine. This causes downward and forward pressure on the ribs, which are rotated so that their anterior ends and the sternum are raised, resulting in a barrel chest. The pulmonary emphysema is marked by a barrel-shaped chest fixed in a rigid inspiratory position, and it is associated with fatigue of the respiratory muscles. This causes deficient ventilation of the lung due to loss of bellows-like action of the chest and failure of the lungs to recoil during expiration because of decreased elasticity of the chest. The dilated air sacs remain full of stagnant air.

A second change occurring in aging people is loss of elasticity of the bronchi, which interferes with the rhythmic motions which normally aid in clearing out secretions and infectious exudates. A third degenerative change in the bronchial tree involves the mucosa, where there may be hypertrophy. Since the bronchial system is used for drainage of secretions as well as for conduction of air to the alveoli, the latter two changes are important because they predispose to inflammation and retard healing. A fourth senile degenerative change which often occurs is pulmonary fibrosis, representing the result of a long series of common respiratory inflammations. It may follow undiagnosed bronchopneumonia and may be of an interstitial type or a peribronchial type. Besides impairing respiratory function, pulmonary fibrosis predisposes to infection.

Thus, in the aging so-called "normal" patient, there are four factors interfering with the adequate ventilation and drainage of the lower respiratory tree. First is senile emphysema, due to vertebral column changes, which fixes the ribs in an inspiratory position with subsequent dilatation of pulmonary alveoli. Besides producing alveolar emphysema, there is a less effective expiratory and tussive effort due to the chest wall being more fixed. (2) Bronchial peristalsis is less effective because of loss of elasticity of the bronchial wall. (3) Hypertrophy of the mucosa narrows the lumen. (4) Pulmonary fibrosis causes a secondary alveolar emphysema, and often chronic adherent pleurisy and bronchitis, as well as predisposing to infection. So, with impaired tussive effort already present in a bronchial system which is impaired in efficiency because of less area for gaseous exchange due to alveolar emphysema,

because of less action in clearing exudates due to decreased bronchial peristalsis, and because of partial blocking of the lumen of the bronchus due to hypertrophy, the lower respiratory tree is barely able to take care of itself under optimum favorable conditions.

If a bacterial infection is superimposed on the above impaired pulmonary apparatus, the result is a suppurative bronchitis. There are added an edema of the bronchial mucosa, secretion of mucus in excess amounts and at times of a very viscous nature, suppuration in the bronchus, and often bronchospasm due to a continued effort to move the mucus out of the bronchi and bronchioles. With the constant bathing of the bronchial mucosa by toxic products of the bacteria, the mucosa remains swollen. The result often is a disease process which is unimproved by ordinary methods of treatment of asthma. In fact, it is not of a truly allergic nature, but consists of an inflammatory process superimposed on a faulty respiratory apparatus which is unable to clear itself of infection. A vicious circle is set up by the bacterial infection, due to defective drainage primarily. (How often an acute maxillary sinusitis which is slow in resolving is rapidly improved after one or two antral irrigations to clear away the thick pus.) The best way to interrupt the circle of the disease process is by bactericidal drugs, by supplementary drainage, and by blocking the inflammatory reaction of the tissue to the bacterial toxins. By decreasing the bacteria and their toxins with antibiotics, the bronchial mucosa is less edematous and swollen. By bronchoscopy, viscous secretions and exudates are removed from the main bronchi. By blocking the inflammatory reaction of the tissues with cortisone or ACTH, the effect of bacteria is offset so that edema of the mucosa is lessened and decreased secretion of mucus takes place.

As to bacterial vaccines, the few cases in which we used autogenous vaccines were complicated by bronchiectasis and were not benefitted. A survey of the literature shows conflicting results with bacterial vaccines. In children, suppurative bronchitis occurring with colds clears up rapidly on symptomatic treatment, or at the most with the aid of the sulfonamides or the antibiotics. Bronchoscopy and cortisone or ACTH are not necessary. In contrast, colds complicated by suppurative bronchitis in older people are often slowly resolved or not completely overcome.

Hence, the oft-repeated statement that asthma in a patient over 40 is of bacterial origin is an oversimplification of the facts because asthma in older patients is due more to degenerative physiologic changes occurring in the aging process than to allergic processes, with the initiating mechanicsm of infection disturbing the balance between fluid exudation and evacuation in the bronchus.

DR. DAVISON: I want to thank Dr. Holinger for his remarks. He brought out quite rightly that children do have this type of disease. It so happens that most of the people who are referred to me for bronchoscopy are adults. Perhaps the pediatricians look for eosinophils; certainly we do. If the infection is very active, there won't be any eosinophils. You will miss the heavy bacterial flora which is a clue to the infection factor unless smears are examined with an oil immersion lens.

I want to thank Dr. Simmons for his remarks. I am glad that he brought out the degenerative changes that can occur. That is why I presented the case of the old lady who had a perfect rehabilitation by having antibiotic therapy.

FURTHER OBSERVATIONS ON THE LOCAL USE OF CORTISONE IN THE NOSE IN ALLERGIC RHINITIS

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THE premise that local application of cortisone in the nose might have beneficial results in cases of allergic rhinitis was considered at some length after observing the excellent results obtained with the topical form of the drug in the treatment of inflammatory eye disease. Therefore, as previously reported,[1] 25 patients were so treated, using a spray of a 1:4 dilution of cortisone* in normal saline to each side of the nose four times daily.

Of the original 25 cases of the series, 12 (or 48 per cent) showed marked improvement; 6 (or 24 per cent) were classified as "doubtful"; and the balance (28 per cent) were considered "failures." Therapy was then discontinued in those patients in whom the results had been classified as "doubtful" or "failure." Of the remaining 12, five continued under treatment and thus have been under observation for more than a year. In addition, 10 new patients have been added to our series.

All of these 10 were carefully screened and have as a common factor the previous treatment of a specific allergy with little benefit. The psychosomatic factor, evident in so many cases of allergic rhinitis, was considered carefully. In an effort to eliminate it as far as possible we were careful not to mention the actual word "cortisone" to the patient, nor did we give any indication that we were hopeful of exceptional results from a new treatment.

Nasal smears were made prior to and during the course of treatment. There was considerable variation in the number of eosinophils found; in some instances there was a great variation in the number both before and during treatment, while in several other cases the eosinophils rapidly disappeared. So variable were the results that we feel this cannot be considered a reliable index to the probabilities of success or failure.

Epithelial cells were either absent or minimal in a number of the preliminary smears, but during the course of therapy an increasing number of these cells frequently was reported. This gives rise to the question of whether or not a continued use of cortisone may induce atrophy of the nasal mucosa. However, to date no subjective or objective evidence of atrophy has been found.

In an effort to evaluate any changes of the mucous membrane, either improvement or atrophy, and to help us in selecting cases for future treatment, biopsy specimens were taken from the middle turbinates, areas of hypertrophy or polypi. These were gone over carefully by our pathologists but with surprisingly meager changes being reported. No actual change in tissue structure was evident and no clues as to the success or failure of the cortisone treatment were obtained. The only finding of

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*Cortone Acetate—Merck & Company, Rahway, N. J.: Cortogen Acetate—Schering Corporation, Bloomfield, N. J.

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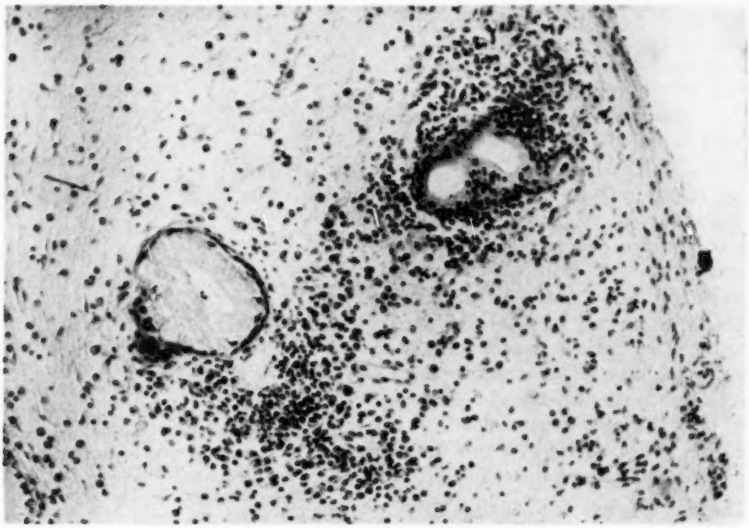

FIG. 1—Section of nasal polyp before cortisone therapy.

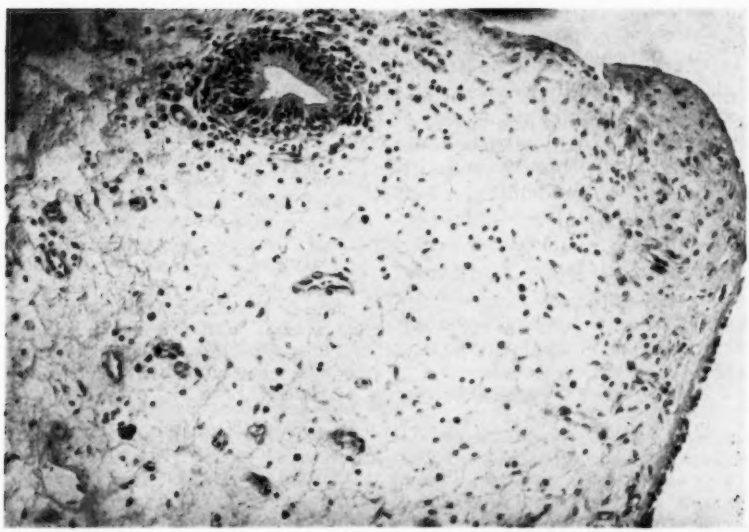

FIG. 2—Section of nasal polyp after cortisone therapy

any significance was a diminution in the number of eosinophils (figs. 1 and 2).

The presence of nasal and sinus infections was determined as far as possible by roentgen examination and such infection eliminated by treatment when indicated. We considered the polymorphs found in the secretions of many of the patients to be, at most, minimal infection in an allergic rhinitis. The cortisone spray had no apparent effect on the infection in one case of acute rhinitis or in one case of acute sinusitis, and in the other patients where acute infection of the nose or sinuses developed during therapy the spray was discontinued until the infection subsided.

Of the entire series, 14 had nasal polypi on initial examination. The effect of the cortisone was negligible on clinical examination, but such subjective improvements as return of the sense of smell and relief of nasal stuffiness were noted as indicative of some shrinkage in the polypi. In one case the polypi disappeared entirely, but any explanation of this can be only conjecture.

No failures have been noted in the second series of 10 patients. Five of these were definitely improved; the others were classified as showing some slight or "doubtful" improvement. This lack of outright failure has raised the question whether or not cortisone has a more marked, or perhaps a synergic, effect on the nasal mucosa in patients having a prolonged course of specific allergic therapy. Or should the result perhaps be considered only a coincidence?

The 3 cases which are presented are representative of the series and show our difficulties and individual results.

Case 1. E.G., white, female, aged 41.

1947

First seen. Had a 7-year history of asthma, urticaria, and marked nasal stuffiness noted with several foods, particularly onions and tomatoes. Nasal polypi had been removed on several occasions. Examination revealed bilateral nasal polypi. Blood count showed 5 per cent eosinophils. Allergic studies were negative for foods; positive reactions were obtained to mold, house dust and bacteria.

1947-48

Allergic therapy given with definite benefit, then discontinued.

1948

Nasal polypi were removed. Pathological diagnosis was "allergic polypi." These polypi contained numerous eosinophils.

4-5-50

Returned to our ENT Clinic with recurrence of symptoms. She had obtained relief of her nasal symptoms and shortness of breath with one of the antihistaminics. Examination revealed a few small polypi, bilaterally; larger polypi in left nostril. Nasal smear showed a few polymorphs but no eosinophils.

Cortisone nasal spray was begun. Six days later she stated she was definitely improved. There was less nasal stuffiness, no sneezing, and nasal discharge, in her estimation, had decreased about 80 per cent. For the first time in two and a half years she was able to distinguish the faint odor of her face powder. On this examination there was no appreciable change noted in the nose.

5-13-50

Slight sneezing. Nasal discharge was more profuse, but sense of smell was good. Nasal smear showed 23 per cent eosinophils and many epithelial cells.

5-27-50

Nasal symptoms had improved with spray but were worse if it was discontinued. She also noted that her nasal symptoms were worse during damp weather, even when using the spray. Examination of the nose showed no material change; nasal smear revealed no eosinophils but many epithelial cells.

6-10-50

Again more marked nasal stuffiness and discharge, but not as great as that present before cortisone therapy. The nasal spray was still producing relief.

6-24-50

Again the nasal stuffiness was worse during a recent cold spell with relief on onset of warmer weather. Nasal smear showed 10 per cent eosinophils. The nasal spray was then discontinued.

11-18-50

Reported a few symptoms during warm weather of summer. Ten days before visit had an acute rhinitis which cleared under treatment. Examination revealed a stuffy nose; polypi had increased in size; cortisone spray was resumed.

12-1-50

Felt improved; breathing better with less discharge; sense of smell absent. Pathologic examination of nasal polypi showed stroma to be edematous with a heavy infiltrate of eosinophils and a few lymphocytes.

12-31-50

Progress satisfactory. Noted certain foods produced sneezing and a stuffy nose. Nasal smear showed no eosinophils.

2-10/3-23-50

Acute rhinitis; spray discontinued.

4-9-51

No evidence of infection; spray resumed.

5-8-51

Less nasal stuffiness, better breathing, slight postnasal discharge, good sense of smell.

6-2-51

Minimal nasal symptoms; slight stuffiness noted at times. Nasal smear showed 52 per cent eosinophils.

Nasal biopsy: loosely structural connective tissue, heavily infiltrated with with eosinophils in some areas. In other areas the infiltrate was sparse and consisted almost entirely of eosinophils.

6-24-51

Nasal smear showed 10 per cent eosinophils; many epithelial cells.

7-7-51

Has improvement when spray is continued. Has good and bad days. Frequently has sneezing and a stuffy nose on arising in the morning.

Note: This is a case with familial history of allergy and a rather long personal history of food allergy, hives, asthma, nasal stuffiness and nasal polypi. Specific allergic therapy and antihistaminics gave relief of symptoms.

Local cortisone spray of the nose produced relief of her nasal symptoms and return of her sense of smell. Symptoms varied during the 15-month period of observation. The eosinophil count of nasal secretions varied even during therapy, polypi were not appreciably altered, and no evidence of nasal atrophy could be detected in the nose.

Case 2. F.P., white, male, aged 58.

1944

Sneezing, nasal stuffiness, excessive nasal and postnasal discharge. Examination revealed a dark right antrum and nasal polypi. Had positive reactions to duck feathers, goose feathers, rabbit hair, giant ragweed, timothy, orchard grass, red top, house dust, alternaria and bacteria. Allergic therapy was started.

1-25-45

Allergic therapy produced slight benefit. X-ray revealed clouding of both antrums. Nasal polypi were noted in both nostrils. Sinus infection cleared under treatment.

3-1-45

Nasal polypi were removed. Allergic therapy resumed and continued to September, 1950.

5-2-50

Complained of stuffiness of left ear. Examination revealed bilateral nasal polypi, particularly on right. No evidence of acute infection found in ear. X-ray of sinuses showed some old thickening of the mucous membrane in both antrums; no evidence of polypi in the sinuses. Cortisone nasal spray was begun.

5-23-50

Nasal polypi were noted to have almost completely disappeared. The patient stated he was more comfortable than at any time for the past two years. Nasal spray was discontinued.

7-8-50

Nasal stuffiness had recurred. Examination revealed presence of nasal polypi which appeared to be about their former size. Spray was resumed.

8-28-50

Patient was comfortable. No polypi found in the nose.

10-4-50

Patient was symptom-free and enthusiastic about the results of treatment. Had been using cortisone spray once daily. Nose was clean; no evidence of polypi; no evidence of nasal atrophy; sinuses were clear on transillumination.

12-13-50

Return of mild nasal symptoms since spray was discontinued, and recurrence of small nasal polypi. Spray resumed.

1-2-51

Again comfortable. No evidence of nasal polypi.

3-15-51

Just recovered from "flu." Small polypi noted in spheno-ethmoid recess. Cortisone continued.

5-8-51

Comfortable. Spray discontinued for one week. Nose appeared clean.

7-2-51

No cortisone for two months. Quite comfortable. Few minute polypi noted in spheno-ethmoid recess.

Note: Cortisone nasal spray produced almost complete relief of nasal symptoms and also

resulted in the disappearance of the nasal polypi. The symptoms and polypi recurred when the solution was discontinued. No evidence of nasal atrophy and no symptoms of atrophic rhinitis were noted although the solution had been used for approximately one year. We have no explanation for the disappearance of the nasal polypi with use of the spray.

Case 3. M.P., white, female, aged 57.

This patient was a markedly psychoneurotic individual with asthma, excessive nasal discharge and nasal stuffiness. Previous therapy, including antihistaminics, produced rather indeterminant relief.

1-25-51

Examination showed markedly pale, edematous mucous membrane and many polypi bilaterally. Nasal smear showed 52 per cent eosinophils and many epithelial cells. Biopsy of nasal polyp showed stroma of polyp was loosely textured and contained acute and chronic inflammatory cells, the eosinophils being very conspicuous. Cortisone nasal spray was begun.

2-6-51

Little relief of nasal symptoms. On nasal examination there appeared to be less edema of the nasal mucous membrane, less discharge and improved airway. Nasal smear showed 55 per cent eosinophils; moderate number of epithelial cells.

2-14-51

Felt improved for past week. Less nasal edema on examination. Nasal smear showed 67 per cent eosinophils; few epithelial cells.

2-28-51

Improvement continued. Nasal examination appeared to substantiate this. Nasal smear showed 26 per cent eosinophils. Biopsy of polyp showed loose myxomatous stroma, infiltrated with a small number of lymphocytes and moderate number of eosinophils.

3-28-51

Improvement continued. Nasal smear showed 1 per cent eosinophils. Biopsy of polyp showed moderate infiltrate of chronic inflammatory cells in a loose edematous stroma. Eosinophils were few.

5-3-51

Symptoms recurred. Nasal congestion rather marked for past week. Tried antihistaminics and cortisone spray without relief. Nasal smear showed 40 per cent eosinophils.

Note: In this patient with rather marked psychoneurotic tendencies at least temporary improvement of nasal symptoms was produced. The cortisone solution reduced the eosinophil content of the nasal polypi and also the nasal secretions. Her therapy was discontinued as we felt she had a temporary and doubtful improvement.

DISCUSSION

Our experiment of using a 1:4 dilution of cortisone in normal saline as a nasal spray has produced a relief of nasal symptoms in approximately 50 per cent of the cases. In addition, 11 patients showed some improvement, but as their therapy was not too satisfactory, we classified the results as "doubtful." We have no explanation for the success of treatment in some cases and failure in others.

The nasal polypi disappeared in only one of 14 cases. This represents a disappointingly small percentage in comparison with the results reported with the use of parenteral ACTH and cortisone. In several patients the polypi appeared smaller on observation, and with this improvement these patients had a return of their sense of smell.

Eosinophils in the nasal smears disappeared most frequently when the cortisone was used locally, but this was not a constant feature. In several instances, such as case 3, the number of eosinophils varied tremendously. At times it was increased, even though the therapy was continuous.

A diminution in the number and nests of eosinophils in a polyp was the only constant finding on biopsy of nasal polypi or mucous membrane. The cellular structure, the various cells, and the amount of edema were not appreciably altered. This was disappointing since it had been hopefully expected that changes would be noted on microscopic examination.

Because of the minimal amount of the drug sprayed into the nose it is doubtful that there is any appreciable absorption of cortisone. Certainly neither a lo-

cal nor general deleterious effect was produced, nor were symptoms or clinical manifestation of nasal atrophy detected, even in those cases under observation while using cortisone for more than a year.

The psychosomatic element was very obvious in one patient, but in all the other cases we felt this factor was absent. Results in each case were carefully evaluated and compared with similar cases receiving other types of allergic therapy.

SUMMARY

Cortisone solution produced local beneficial results in approximately 50 per cent of the patients. Improvement was present while therapy was continued, but symptoms returned a short time after spray was discontinued. The longer the period of use, the longer the period of relief. No explanation from either clinical observation or pathologic tissue examination can be offered as to why benefit is obtained in some cases while treatment in other cases results in failure. No nasal atrophy was produced in any of the 35 patients, some of whom had continued therapy for over a year. We feel that while cortisone nasal spray can produce relief of nasal symptoms in some cases, it should not replace specific allergic therapy.

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THE UVEAL TRACT AND RETINA: CONSIDERATION OF CERTAIN EXPERIMENTAL AND CLINICAL CONCEPTS

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I. INTRODUCTION

IN this discussion, attention is centered on immunity and allergy as they affect the uveal tract. Although the retina alone may be diseased in certain cases, physiopathology of the retina is usually secondary to choroidal disease. Many concepts of the physiopathology of chronic uveal inflammation are based on animal experimentation, much of which, especially that concerning tuberculosis and syphilis, is applicable to man. This is also true of chronic "focal infection" but to a lesser degree.

II. CLASSIFICATION OF ALLERGIC UVEAL MANIFESTATIONS

For many years, the terms "serous" and "plastic" have been used to designate different types of iritis and iridocyclitis. Recently, Woods applied the name "granulomatous" to the plastic type and "nongranulomatous" to the serous type of inflammation.[99,100]

Nongranulomatous Uveitis

In the serous or nongranulomatous type, the inflammatory reaction is considered to be largely caused by bacterial hypersensitivity, which may be due to toxins and other bacterial products or to the bacteria themselves. It may affect ocular tissues alone or may be associated with involvement of other organs. Such hypersensitive reactions have an acute onset and usually are associated with marked circumcorneal congestion. Although there may be gelatinous or fibrinous exudates in the aqueous, keratic precipitates, as a rule, are small. Recovery is commonly rapid and complete with few inflammatory complications, unless repeated attacks occur. Usually, vitreous exudates are not numerous and, although there may be some retinal or subretinal edema, no masses or exudates are found.

Granulomatous Uveitis

In the plastic or granulomatous type of uveitis, the original hypersensitivity of the uveal tissue is produced by micro-organisms of low virulence, and when the living organisms again invade the uveal tissues, a mild inflammation results. This reaction produces the plastic type of iritis typified by the appearance of large, "mutton fat" precipitates and iris nodules, which were first described by Koeppe. Exudates having a greasy, grayish appearance are often

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observed on the anterior capsule of the lens and are commonly associated with posterior synechias. The Koeppe nodules and the keratic precipitates are composed largely of epithelioid cells. In tuberculosis, the epithelioid cells are stimulated by the lipoid fraction of the tubercle bacillus.[86] Vitreous opacities are characteristically heavy and numerous and often interfere materially with vision. When the retina can be seen, there are massive exudates associated with subretinal edema.

Granulomatous uveitis may be produced by syphilis, tuberculosis, brucellosis, sarcoidosis, lymphogranuloma venereum and toxoplasmosis. However, viral and streptococcal infections, when the organisms are not too virulent, may also cause these lesions. Some investigators claim that the organisms actually live and proliferate in these lesions and give rise to a characteristic reaction because of the high degree of local hypersensitivity of the tissues. The natural and acquired resistance of the individual produces compensatory overgrowth of granulomatous tissue and frequently tissue destruction and caseation necrosis. In uveal tuberculosis, the caseation necrosis is the effect of the protein fraction of the bacillus.[82] If immunity is high and tissue hypersensitivity is low, the lesions are limited in extent and recurrences are rare. If immunity is low, as it often is in young persons, the lesions are severe and tend to spread rapidly. Later in life, if hypersensitivity becomes marked, the lesions may be much larger and progress to caseation and sometimes to severe destruction of the tissues.

III. ROLE OF ALLERGY IN CERTAIN SPECIFIC CONDITIONS AFFECTING THE RETINA AND UVEAL TRACT

1. *Allergic Manifestations of Focal Infection in Lesions of the Retina and Uveal Tract*

Focal infection is discussed first, even though it is the most controversial subject and many of the conclusions are debatable.

Billings in America in 1912[18] and Lang in England in 1913[59] first drew attention to the important rôle played by a localized focus of infection in producing organic disease. The influence of these infections on the eye is fairly universally admitted, although the mechanism of the action is still debated. There is a great deal of experimental and clinical evidence that living micro-organisms may be carried by the blood stream to the eye and produce inflammation. Tuberculous material was inoculated into guinea pigs by Cohnheim in 1867 and tubercles were found in the uveal tract when the eyes were examined pathologically. Spirochetes were found in the cornea by Igersheimer in 1922 when he produced metastatic interstitial keratitis experimentally. Pneumococci and streptococci cultured from foci of infection, when injected intravenously into rabbits, were recovered from subsequent uveal lesions by culture and were demonstrated in microscopic sections of these eyes. The first of this work was done by Rosenow.[80] Much less is known concerning the rôle of virus infections in producing endo-ocular inflammations. However, it seems quite likely that herpetic iritis is of viral origin. Friedenwald and McKee[41] produced uveitis in animals with the filtered spinal fluid from a patient who had bilateral uveitis.

On the other hand, no pleuropneumonia-like organisms or other filterable agents were isolated from the aqueous or filtered nasopharyngeal washings of 16 patients with inflammatory eye diseases.[8] No virus was cultured from the exciting eye of a patient with sympathetic ophthalmitis but mildly toxic staphylococci were grown from the choroid.

Bacterial filtrates have been accepted as agents in producing ocular inflammation since Guillery reported his findings in 1911.[46,47] Staphylococcus toxins were associated with ocular inflammation as early as 1903 by Stock.[88] More recently, Berens and Nilson[12] reported the occurrence of iritis in rabbits' eyes following the intravenous injection of filtrates of coliform bacteria. Even though bacteria may sometimes be cultured from the aqueous of patients' eyes as shown by Berens et al[8] and by Verrey,[94] it is possible that some other mechanism may be responsible for the production of uveal inflammation.

That uveal inflammations may be on an anaphylactic basis is probably true. It was demonstrated years ago by Elschnig, Sattler and others that an injection of foreign serum into the eye of an experimental animal, followed by an intravenous injection of this serum, resulted in ocular inflammation of an exudative type. The most recent work in the field was done by Foss,[38] who demonstrated that injections of foreign proteins (egg-albumin, horse serum) into the vitreous of rabbits, were followed by the appearance of a positive serum precipitin test in the blood stream on the fifth and sixth day and iridocyclitis within 8 to 13 days. That bacterial toxins can produce a similar reaction has been confirmed recently by Ayo,[2,3] who worked with culture filtrates of Escherichia coli, which produced the Shwartzman phenomenon. Increased protein content was found in the aqueous associated with hyperemia, which appeared a few minutes after the injection and persisted for 24 hours. This type of response to toxins may account for some cases of iritis which are of unusually short duration. However, the most common chronic ocular inflammations seem to be associated with some other mechanism which is considered allergic in nature and this subject will now be discussed.

Ophthalmologists are deeply interested in recurring attacks of iritis, iridocyclitis and chorioretinitis because of the high incidence of blindness which results from these inflammations.[32] That the tissues of the eyes of experimental animals can become sensitized to bacteria, either by repeated injections of the organism or with agar implants, has been known since the work of Derick and Swift in 1928.[33] These facts have been confirmed by Brown[19] and others.[8] Even Corynebacterium xerose, which is ordinarily considered nonpathogenic, can produce pathologic effects. The association of iritis with sensitization seems to be borne out by the fact that long after the specific antigen can no longer be found in the aqueous, the lesion may be reactivated by an intravenous injection and the aqueous again contain the specific protein.[83] In working with uveitis in relation to arthritis,[8] it was noted that sometimes an organism (alpha hemolytic streptococci and beta hemolytic streptococci) would not produce intraocular inflammation following a single intravenous injection, even though the bacteria were present in the choroidal vessels of the animals. However, inflammatory lesions were produced by repeated injections of the living organisms but not with filtrates, or with the carbohydrate or protein fractions of these bacteria. Uveitis in animal eyes resulted from the intravenous injection of cultures of living streptococci in animal eyes previously "immunized" with formalin-killed streptococci.[15]

In spite of the lack of absolute proof of the theory of focal infection in man, it seems likely that there is a group of endogenous ocular inflammations produced by sensitization of the uveal tract or other ocular tissues to relatively innocuous organisms, which may later be activated by bacterial products or by the bacteria themselves.

The great variation in the proportion of cases of uveitis attributed to the different infections by various authors indicates the complexity of this differential diagnostic situation. For example, in France, syphilitic treatment is administered for uveal lesions, even when little evidence of syphilis exists. In Germany, uveitis patients are more often treated for tuberculosis; whereas in England, the foci of infection are usually considered as causal. In the United States, some groups remove or treat foci of infection, others prescribe for syphilis and tuberculosis and many merely treat symptoms and administer antibiotics and sulfonamides, usually without prior bacteriologic investigation.

This uncertainty concerning the etiology of most uveal inflammations makes it imperative that we continue to study this important subject, especially in relation to allergy. Some of the authors who formerly attributed much uveitis to focal infection now consider this to be a minor factor. The same is true of focal infection in relation to arthritis. We question the statements by some of these authors who discount focal infection in the etiology of disease because they claim to have removed "all foci" and that the disease treated was unaffected. From our personal experience, removal of infection is often impossible in patients who have chronic sinusitis, and sometimes problematical in oral infections, to cite only two examples. On the other hand, it is important to continue research on viruses as possible primary etiologic factors.

2. Ocular Syphilis

Although it is believed that allergy plays an important part in the ocular lesions associated with syphilis, we are greatly handicapped in this type of research by the fact that there are no clinical tests which will demonstrate, with certainty, hypersensitivity of the syphilitic patient. For example, the degree of allergy of the subject and his immunity are not indicated by complement fixation, precipitin or Hecht's tests. For many years, research workers have attempted to prepare an extract of treponema, which might serve the same purpose in syphilis as tuberculin does in determining vaccinal hypersensitivity in tuberculosis. Although such a preparation was developed by Noguchi and named "luetine," it was soon found that the skin of syphilitics was generally more sensitive than the skin of nonsyphilitic patients and false positive reactions with nonspecific products were often observed. Furthermore, a positive reaction occurred in healthy subjects after the administration of potassium iodide. In addition, doubt was cast on the treponema that Noguchi used in producing syphilis. Therefore, in determining the part played by allergy in the syphilitic patient, it is necessary to attempt to draw conclusions from experimental and clinical studies and from anatomopathologic material.

In observing experimental allergic manifestations of ocular syphilis, it has been noted by means of reinoculations that ocular immunity to the organism occurs much later than in the other tissues of the body. Furthermore, one cannot always obtain general immunity by inoculating the cornea or the anterior chamber with treponema, possibly due to the lack of blood vessels in the cornea.

Allergic syphilitic lesions of the uveal tract are considered mainly from the standpoint of insufficiently treated patients and inflammation associated with gummatous lesions of other parts of the body. It is well known that the uveal tract and the central nervous system are slow to acquire immunity to syphilis. Therefore, when a patient is being insufficiently treated, the blood stream may produce a Herxheimer reaction, which is considered by many to be an

allergic phenomenon. In this instance, the sensitized uveal tract, which is not immunized, could be the seat of inflammation.

There are two reactions associated with secondary and tertiary syphilis which are considered allergic by syphilologists. One is a fibrous reaction with mild inflammatory lesions affecting the parenchymatous tissues of the nervous and cardiovascular systems. The other is a gummatous inflammation characterized by a marked tissue reaction in which a spirochete is found occasionally.

Gummas are believed to be due to tissue hypersensitivity to treponema. The iritis and uveitis observed in late syphilis and the fact that they are present in patients who have other gummatous lesions are anatomopathologic arguments in favor of considering the ocular lesions as allergic phenomena. As an example of allergy, Gutmann[48] referred to a case in which a large gumma of the sclera followed a subconjunctival injection of sodium chloride and was subsequently cured by antisyphilitic treatment.

3. *Ocular Tuberculosis*

Our concepts of tuberculosis, including ocular tuberculosis, have changed considerably since the discovery of the organism by Koch and his early classic description of the disease. His original ideas have been superseded by the findings that the organism is pleomorphic, can exist in the form of a filterable virus and, under certain conditions, can lose its resistance to acids. From the standpoint of anatomy and clinical classification, Ranke[74] proposed certain subdivisions which were quite generally accepted at the time. The first stage corresponds to the primary infection in the tracheobronchial lymphatic glands and, more rarely, in the region of the intestines. The second stage is characterized by hypersensitivity and the tendency for rapid extension of the lesion. The third, or adult, stage is that of resistance of the host to the tubercle bacillus. Minet and Warembourg[66] have added a new concept. They believe that a tuberculous patient, whether affected with a latent or frank infection, may alternate a state of hypersensitivity with that of resistance, thus exposing the patient to the hazards of infection and reinfection, whether exogenous or endogenous. The tissue reactions may be different because of various interactions between the micro-organisms and the host. Ideas have also changed concerning the significance of the cutaneous test. Today, a negative dermal or intradermal reaction is not conclusive proof that the patient has never been infected by the tubercle bacillus.

There are also new concepts with regard to immunology. For example, one organ may acquire immunity before another. In this way, a cicatrizing pulmonary lesion may liberate products of disintegration into the blood stream and produce an inflammation in an eye which was sensitized but not yet immunized.[58] It is also recognized that patients with ocular tuberculosis may have a special form of miliary tuberculosis named "discrete miliary tuberculosis" or "interstitial pulmonary tuberculosis" or "dry pleuritis." This type of lesion has a tendency to disseminate bacilli into the blood stream.[22] The rôle of bacterial products has been used to explain tuberculous interstitial keratitis, phlyctenular keratitis, certain forms of uveitis and recurring forms of vitreous hemorrhage in adolescents. Unfortunately, no one has been able to isolate the substance which produces the bacterial hypersensitivity.

There can be little doubt that allergy and immunity play an important rôle in tuberculosis affecting the uveal tract. From the experimental standpoint, the ocular lesions are quite comparable to the classic descriptions of Koch. If ba-

cilli are injected into the eye of a healthy animal, a slowly developing process results in the formation of tubercles containing bacilli. If the animal has tuberculosis before the injection is given, the eye reacts to the inoculation of the bacilli by the production of inflammatory lesions, frequently caseous but containing few bacilli. It is well to remember, as Rich and MacCordock have pointed out, that the character of the tuberculous lesions is determined by the number and virulence of the invading microorganisms and by the hypersensitivity of the tissues invaded. This process is modified by the resistance of the organism.

4. *Recurrent Hemorrhages of the Vitreous in Adolescents*

Some recurrent vitreous hemorrhages are thought to be caused by hypersensitivity to tubercle bacilli. The typical appearance of tuberculous periphlebitis has been produced in animals by inoculation with tubercle bacilli and later hemorrhages have developed in the vitreous following the intravenous injection of small doses of tuberculin.

It has been shown by the work of Rich[75] that allergy and immunity in tuberculosis can be separated and that, at least experimentally, allergy can be abolished and immunity retained. That cells can become hypersensitive even without nervous or circulatory influences has been demonstrated by Rich and Lewis[76] and others.[1,67] Cells in explants originating from a tuberculous body were sensitive and damaged by the addition of tuberculin; explants from nontuberculous animals were not damaged when tuberculin was added to the culture medium. It is generally believed that this cytotoxic selective effect of tuberculin on cells from tuberculous animals is the result of an antigen-antibody reaction. Such a mechanism may also be a factor in the production of retinal hemorrhages.

A patient with recurring vitreous hemorrhages, seen by one of us (CB), responded poorly to treatment which included the removal of foci of infection, tuberculin desensitization and the administration of an autogenous vaccine consisting of alpha hemolytic streptococci (throat), nonhemolytic streptococci (throat), Staphylococcus aureus (throat) and nonhemolytic enterococci (postnasal). Dr. Eugene Blake, who referred this patient, stated that the patient later improved rapidly following the administration of moccasin snake venom.

5. *Sympathetic Ophthalmitis*

We believe that hypersensitivity to uveal pigment plays some part in the development of sympathetic ophthalmitis. Whether this is a primary or secondary rôle is not clear. Our impression is that a more important factor is the sensitization of the patient to bacterial products and that this is more likely to be the exciting factor producing the ocular pathology. The occurrence of organ-specific hypersensitivity to uveal pigment may be a factor in preparing the fellow eye for ocular infection or for the possible somewhat selective effect of bacterial products from other foci of infection. Although a virus may be the exciting agent, we have never succeeded in culturing one from an eye with sympathetic uveitis. A staphylococcus of low virulence was cultured from one eye with sympathetic ophthalmitis when using the most careful technic to avoid the possibility of contamination. In the few cases we have seen which were diagnosed as sympathetic ophthalmitis, a coexisting disease has always been present in the nasal accessory sinuses or some other focus of infection. The best results occurred in those cases in which the infected sinuses or other foci could be properly treated. Bacterial antigen desensitization seemed to be

helpful in certain cases. The following case is illustrative of this statement.

Case 1: W.P., white Italian male, 26 years of age. A piece of steel had penetrated his left eye in 1934 and a magnet extraction had been attempted unsuccessfully. When examined in March 1938, vision O.D. was 20/15 and O.S., light perception but no projection.

Roentgenologic examination revealed a foreign body located 8.5 mm. behind the center of the cornea. It was impossible to remove it and a combined extracapsular extraction was performed. The patient was discharged from the hospital with the diagnosis of detached retina and retinitis proliferans. Enucleation was refused.

In March 1943, the patient reported that he had received a blow on the left eye. In October 1943, the patient noticed blurred vision in his right eye. Vision was 20/30 and the near point of accommodation was remote. In November, the vision was more blurred with attacks of poor vision and photophobia. Small keratic precipitates and an aqueous beam were seen with the biomicroscope in his better eye. Enucleation of the left eye was performed in November 1943, the optic nerve being cut 15 mm. behind the eyeball.

The histologic study of the eye by Theobald, Samuels and Wilder resulted in the diagnosis of sympathetic ophthalmitis.

In an effort to protect his right eye, several consultations were ordered. The rhinologist found polypoid changes in the right ethmoid sinus and the dentist reported small cavities in one molar tooth. The patient was hospitalized. The eye became hypotonic and vision was reduced to 20/50-2. Cells in the aqueous, marked aqueous flare and a congested disc were present. Therapy included local nasal treatment, intravenous typhoid injections, neosalvarsan, repeated intravenous injections of a 10 per cent solution of sodium salicylate, injection of crude liver extract and the use of atropine locally. A small polyp was removed from his right ethmoid region.

At the end of December, the patient was discharged from the hospital with no aqueous beam, some pigment deposits on the anterior surface of the lens, only a few cells in the aqueous and a normal fundus. Toxic staphylococci were found in nasal cultures and an autogenous vaccine was prepared and administered. In February, the vision and the near point had returned to normal. A physical examination including most of the accepted laboratory tests for chronic infection, revealed no pathology with the exception of a positive patch test for tuberculosis.

We have found it difficult to evaluate intradermal tests in determining sensitivity to uveal pigment in sympathetic ophthalmitis and the results in 82 patients with inflammatory eye diseases are shown in table I.

Discussion of table 1: Of the 9 patients with positive tests, 6 had chronic uveitis, probably from a constitutional cause and without evidence of sympathetic ophthalmitis, 2 had cataract and 1 had keratitis. Three patients had conditions which were diagnosed clinically as sympathetic ophthalmitis but only one was verified histologically. The intradermal tests with uveal pigment were negative in all three of these patients, even though we followed Friedenwald's suggestion of excising the skin on the fourteenth day following the injection of uveal pigment. This is apparently an excellent method of differentiating a positive from a negative reaction, as previously reported.[4] In one case of expulsive hemorrhage with incarceration of uveal tissue in the wound, a mild accidental infection occurred in the skin and a histopathologic diagnosis of the reaction to pigment could not be made. This reaction might have been considered positive from the clinical standpoint and demonstrates the importance of examining the excised tissue histologically.

6. *Hypertension in the Central Artery of the Retina in Allergic Conditions*

Gutmann[49] examined 10 patients suffering from allergy and found not only increased blood pressure in the humeral artery but also that 8 of the 10 patients had a systolic blood pressure greater than 100 mm. of mercury in the central artery of the retina. The normal pressure in the artery is approximately 40 mm. Hg.

TABLE I

RESULTS OF 82 UVEAL PIGMENT TESTS IN VARIOUS EYE DISEASES

DIAGNOSIS	POS.	NEG.	DOUBTFUL
Uveitis due to constitutional causes	6	16	5
Contusions with traumatic uveitis:			
No sympathetic irritation		3	
With sympathetic irritation			1
Endophthalmitis phaco-anaphylactica (One eye lost after lens extraction)			1
Penetrating wounds involving uveal tract:			
Recovery without enucleation: no sympathetic ophthalmitis		16	1
Enucleation of injured eye; no sympathetic ophthalmitis		3	1
Sympathetic ophthalmitis:			
Foreign body, not sympathetic pathologically		1	
Penetrating wound not sympathetic pathologically		1	
Postoperative case enucleation, cultures negative, pathologic report sympathetic ophthalmitis		1	
Buphthalmos		1	
Optic atrophy		1	
Cataract	2	14	4
Keratitis	1	1	
Normal eyes		2	
Total of Cases	9	60	13

The author then sensitized 5 rabbits with horse serum injected into the marginal vein of the ear. The retinal systolic pressure before the injections averaged 62 mm., but 14 to 18 days following the injections, it was as high as 100 mm. in one rabbit and the lowest was 71 mm. No mention is made of any generalized manifestation of sensitization in the rabbits. Coca and others[28,60,65,72] have reported several cases in humans, in which vascular hypertension was controlled by elimination of known allergens.

7. *Participation of the Retina in Ocular Allergy to Light*

According to Lehrfeld,[60] ocular allergy to light exists principally as a physical allergy related to solar and dermographic urticaria, but it is possible that in certain pathologic states the visual pathways are rendered sensitive to light. Tolerance to light depends not only on the amount of pigment in the eye and on the sensitiveness of the light-receiving and light-conducting apparatus but also on the light composition and its mirrored reflection on the cornea and lens. Experiments have shown that in cases of intolerance to light all subjects experienced a greater degree of comfortable vision, and over a longer period of time, with tinted lenses than with white. It is concluded that tinted lenses of a color pleasing to the eyes and with uniform transmission of all visible wave lengths will reduce intolerance, maintain a maximum visual acuity and subdue actual physical light allergy.

8. *Retinal Separation and Allergy*

Lowenstein[62] reported 4 cases of retinal separation associated with allergy. The first case suggested that the angioneurotic retinopathy was probably of allergic origin. The second case was diagnosed as one of posterior scleritis with exudation both in the orbital and subretinal tissues; there was no exophthalmos but there was a limitation of eye movements with submacular or intramacular exudation. The third case showed a central fundus lesion, similar

to that of the second case, associated with a typical tuberculous iridocyclitis. The fourth case was one of conjunctival chemosis and retinal detachment in a myopic eye. The allergic inflammatory reaction was fully described by Lowenstein.

According to Prewitt,[71] an allergic swelling in the choroid may be a factor in retinal detachment. A man, aged 62 years, had sudden loss of vision and the appearance of corrugations in the fundus, associated with nodular swellings on the body. These recurred on numerous occasions before the retina became completely detached. Several findings suggested an allergic basis. The appearance of blebs on the cornea coincident with a severe allergic dermatitis and their simultaneous subsidence, seemed to indicate that an allergic reaction may become localized on or within the eyeball.

Food has also been implicated in retinal allergy. Plumer[70] saw a man, aged 35 years, with a definite allergic diathesis. Transient edematous retinal lesions were observed at the macula. No attacks have occurred following total abstinence from certain foods, indicating food allergy as the likely etiologic basis. One of us (LJG), in consultation with Dr. Arthur Coca, observed a case that demonstrated a retinal edema on repeated occasions following the ingestion of buckwheat. Another patient, seen by one of us (CB), a man aged 36 years, developed edema of the right macula and paresis of convergence associated with giant urticaria 30 minutes after eating mackerel.

We have observed several patients with unilateral sinusitis associated with homolateral detachment of the retina. The following case reports are examples.

Case 2: Miss C.J., aged 23, had a serous detachment without tear and was operated upon three times in 1924 by means of trephine and suction, with a recurrence each time. Two months after exenteration of the ethmoids and desensitization with an autogenous vaccine, trephine and suction was repeated and complete reattachment resulted. As the eye revealed no active inflammation, the condition was considered to be on an allergic basis.

In case 3 the retinal detachment was apparently responsive to the treatment of focal infection.

Case 3: Mr. J. T., aged 56, was referred by Dr. Eugene Blake for the treatment of incipient retinal detachment in his left eye. The patient had lost the vision in his right eye from retinal detachment 10 years previously. The left eye showed a flat detachment in the lower nasal quadrant and two retinal holes. Since the right eye had not responded to surgery, the patient was admitted to the Focal Infection Clinic of the New York Eye and Ear Infirmary, where a complete examination for chronic infections was performed. Following extraction of two infected teeth and sphenoidectomy on the left side, the retinal detachment subsided and the holes were observed to heal spontaneously. The patient was discharged three weeks after admission with a full visual field in the affected eye.

9. *Central Angiospastic Retinopathy*

Central angiospastic retinopathy usually occurs in young adults. There is a slightly raised reddish area of preretinal edema, surrounded by a ring-shaped light reflex and reduced visual acuity. Vascular instability in such cases was demonstrated first by Horniker.[54] Later, Gifford and Marquardt[43] confirmed his findings and demonstrated the importance of tobacco in causing peripheral vascular spasm. In one case, smoking one cigarette caused the peripheral temperature to fall approximately 8 degrees F. Several authors[34,96] consider allergy as a likely etiologic factor in peripheral vascular disease. Retinal lesions resemble urticarial wheals and their improvement with vasodilation[35] and hyperventilation through forced respiration, as practiced by Bietti, suggests angiospasm and secondary anoxia as important factors in the etiology. The

debilitating effects of fatigue, tobacco and emotional stress must not be overlooked. However, no common physical findings were noted in 176 cases, reported by Cordes and Aiken,[31] which they named "foveo-macular retinitis." Amebiasis seemed to be a likely factor in a man, 46 years of age, who was treated with cortisone and vasodilators with some improvement, but it was not until the parasites were recovered from his stool and treatment instituted for amebiasis that vision rose from 20/200 to 20/30 and the central scotoma became barely perceptible.

10. *Allergy and Retinal Lesions Associated with Acute Disseminated Lupus Erythematosus*

Allergy has been seriously considered as a likely cause of lesions associated with acute disseminated lupus erythematosus. Some authors consider the intraocular lesion as an allergic response to tubercle bacillus toxin.[17,69] According to Stillians,[87] Brocq's theory that toxins from infectious foci of various kinds are responsible for the lesions of lupus erythematosus has long been favored by dermatologists. That such lesions are the result of "infectious allergy" is the view held by Stokes, Beerman and Ingraham.[89] Cordes and Aiken[31] consider flame-shaped retinal hemorrhages, cotton wool exudates and papilledema or "true retinitis," the most typical and frequent fundus picture associated with acute disseminated lupus erythematosus. These authors discussed the allergic and other theories of the cause of the retinal lesions.

11. *Allergy and Acute Macular Exudative Chorioretinitis*

According to Duggan,[35] most patients having acute exudative choroiditis are benefited by vasodilation therapy because this condition is the result of allergy. He believes that histamine sensitivity is a factor.

12. *Probable Association of Allergy and Glaucoma in Uveitis and Possible Association of Chronic Primary Glaucoma with Allergy*

Secondary glaucoma: Because allergy seems to be closely associated with uveitis caused by infection (e.g., tuberculosis, syphilis and probably "focal" chronic infections), the glaucoma secondary to the uveal process is certainly partially attributable to the allergic state of the ocular tissue. The difficulty encountered in making an exact etiologic diagnosis has been discussed.

Chronic primary glaucoma: That approximately 10 per cent of the cases diagnosed as chronic primary glaucoma are definitely inflammatory, at least those seen in the United States, is brought out clearly by pathologic studies.[8] Although the association of so-called true primary glaucoma with an allergen is by no means clear, it has long been the belief of one of us (CB) that such a relation does exist and that allergy[10,13] to both foods and to bacteria probably plays a part in the disease.

In almost all patients with chronic primary glaucoma, some chronic infection can be discovered by diligent search,[5] but as this is also true of many patients in the age group in which "primary" glaucoma is found but who do not have glaucoma, this fact cannot be used as proof of an etiologic relationship. Furthermore, attempts to produce an entity similar to "primary glaucoma" in human eyes by establishing chronic infection in animals has been unsuccessful in the hands of one of us (CB).[13]

It has long been our belief that exacerbations of chronic sinusitis are often associated with attacks of ocular hypertension and that the irides of eyes operated upon for primary glaucoma usually show a mild inflammatory reaction in contradistinction to eyes operated upon for congenital anomalies. For this reason, 53 cases of chronic primary

glaucoma were studied in relation to sinus disease.[13] Thirty-seven of these patients (70 per cent) had either unilateral glaucoma or showed more serious signs of glaucoma in one eye than in the other. In each of these 37 patients, the nasal involvement was greater on the same side as the eye with the more active glaucoma. Even more significant is the fact that in no case was there marked nasal pathology on one side associated with a markedly active chronic primary glaucoma on the other side. As was expected, Woods,[101] in his discussion, took exception to this suggestion. It is regrettable that most references concerning sinus disease as a possible etiologic factor in primary glaucoma are not documented by case reports.[37,61,63,79,90] However, according to Epshteyn,[36] Spector reported that in 71 of 76 cases of bilateral glaucoma, roentgenograms revealed greater cloudiness of the sinuses on the side on which the disease was most advanced.

If we concede, for the moment, that the data on unilaterality of sinus disease and primary glaucoma is at least suggestive of some possible relationship, it is still not clear how the eye is affected. That the eye on the more affected side is likely to be more congested and that the nervous impulses are more likely to affect the circulation on that side, has been discussed.[13]

The probable importance of allergy in this relationship has been reported.[13] We still believe from our clinical and research experience and from the experience of others[12,20,21,30,64] that many of the patients are sensitized to their own bacterial toxins or products and that the ocular allergy engendered is responsible for much of the disease we name primary glaucoma. The improvement of the glaucoma in many patients following nasal treatment or surgery and the use of autogenous bacterial antigens is to us highly suggestive. Another point to be pondered is that allergy, when it affects the nose and nasal sinuses, may affect the trachea, bronchi, labyrinth and even the cochlea.[50]

It is important from the standpoint of this discussion to cite Goodale,[44] who in 1916 pointed out that bacteria played a part in the etiology of allergy of the upper respiratory tract. This report was followed by similar reports by Cooke[29] and later by Selfridge[84] and Hansel.[51] More recently, Kistner (1929),[57] Grove and Cooke (1933)[45] and Semenov (1938)[85] have re-emphasized the part played by bacteria. However, as recently as 1951,[50] Hansel stated, "The significance of bacteria in allergy is still a moot question. Bacterial allergy has not been definitely proved. Bacteriologic investigations have not clearly distinguished between saprophytic and pure suppurative or pyogenic infection."

Because of the controversial nature of the subjects of primary glaucoma, focal infection and allergy and their possible relationship, one is tempted to lengthen this discussion but it is necessary to pass on to food allergy and glaucoma.

Our interest in bacterial allergy, as a possible factor in glaucoma, and our attempts to desensitize with autogenous antigens led us to study the question of food allergies and glaucoma. In our experience, the usual means of determining the allergens responsible for food allergy have been disappointing.

Since our attention was drawn to the pulse-diet method of Coca,[28] we have reported[10] 3 cases of chronic primary glaucoma which seem to have been benefited by the elimination of food allergens. The usual treatment with miotics, nasal therapy and attempted desensitization with autogenous bacterial antigens had been supplemented by the removal from their diets of food allergens, as determined by the pulse-diet method. In one case, the hitherto uncon-

trolled tension was brought under control apparently only after the institution of a diet free from the offending allergens. In the other two cases, surgery and medical treatment controlled the glaucomatous hypertension but failed to check progressive loss of visual fields. The fields showed marked improvement after the institution of an "allergen-free diet." Although it seems possible that primary glaucoma could be an expression of chronic angioneurotic edema on an allergic basis, and the histologic findings of edema by Theobald[91] suggest this possibility, the lack of satisfactory response by chronic cases to ACTH and cortisone is a point against this concept.[9] However, several men have reported good results in glaucoma with adrenal gland preparations.[52,55,56,102]

Because of the many anatomic, physiologic and pathologic factors involved in primary glaucoma and the great importance of psychosomatic factors, our findings in patients with sinusitis, other "focal" infections and food allergy must be examined most critically. However, from our experience, the question of bacterial and food allergy should be considered in patients who have chronic primary glaucoma and any allergic condition should be treated as a local manifestation of a systemic disturbance. In many patients we must assume the responsibility of making a differential diagnosis, keeping in mind that secondary infection frequently complicates allergy and that the reverse may be true.

One of us (CB) has observed several patients whose ocular sensitivity to drugs (e.g., atropine, hyoscine, pontocaine) seems to have been relieved after drainage of an infected sinus.

Case 4:Rev. A.R.M., aged 58, had an iridocylitis of one year's duration following an intracapsular cataract extraction. The iridocylitis developed two weeks after the onset of a severe upper respiratory infection. Allergic blepharoconjunctivitis followed the use of all the commonly prescribed mydriatics, cycloplegics and local anesthetics. The allergic blepharoconjunctivitis and iridocyclitis persisted for one year until a small infected ethmoid cell on the side of the ocular inflammation was opened and drained. Following this procedure, cycloplegics, mydriatics and local anesthetics no longer produced a blepharoconjunctivitis.

Another moot subject is whether acute congestive glaucoma may be an allergic manifestation in certain cases. That there is some type of ocular vascular crisis in patients who have an acute attack of congestive glaucoma is generally conceded. The cause or causes for the vascular changes in the ciliary body are still controversial. We have long believed that allergy to bacterial toxins might be a factor because the sedimentation rate has usually been elevated in these patients. The marked skin hypersensitivity to the intradermal injections of the aqueous from eyes affected with acute glaucoma and the lack of skin response to aqueous from normal eyes and to physiologic saline solutions led Friedenwald[39] to conclude that "in some cases of acute glaucoma there is a transportable vasotoxic substance demonstrably present in the intraocular fluids. Whether this is the cause or result of glaucoma cannot at present be determined." Later, Friedenwald and Pierce[42] injected histamine into the aqueous and vitreous. Both injections were accompanied by a rise in tension. In most cases of acute glaucoma seen by us, emotional factors could not be eliminated. Whether emotion causes vasoconstriction and anoxia directly followed by edema, or indirectly through the effect of the increase of epinephrine content of the blood and resulting prolonged vasoconstriction, is still a moot question. That vasoconstriction may be a factor is suggested by the favorable response to amyl nitrite in acute glaucoma, reported by Rabinowitch,[73] because this drug acts directly on the blood vessels. From these and other consid-

erations, it seems obvious that in some cases allergy may be responsible for the increased capillary permeability and vasospasm, that we can at least postulate as occurring in acute glaucoma. However, we cannot doubt that other etiologic factors may be active.

IV. DIAGNOSIS OF VARIOUS TYPES OF ALLERGY AFFECTING THE RETINA AND UVEAL TRACT

Because the clinical diagnosis of uveal lesions is much more difficult when the cause of the inflammation is tissue allergy and not infection by organisms, this subject requires special consideration. Hypersensitivity to organisms must be demonstrated; but in the eye this may be dangerous and, therefore, dermal tests are used. Even though other causes are excluded and there is evidence of past recent infection, the tests at our disposal are usually inconclusive and the diagnosis is made largely on clinical experience rather than by incontrovertible scientific proof. The difficulty of evaluating skin tests is increased by the fact that persons are exposed almost constantly to the organisms which are likely to cause uveitis, as streptococci, staphylococci and tubercle bacilli. Furthermore, other organisms, for example, coliform bacteria, may require consideration, even though conclusive evidence of implication in uveal disturbances is lacking. These organisms[12] have been found in the upper respiratory tract of a large percentage of patients with serious inflammatory eye diseases and whose skin usually reacted strongly to these bacteria.

Moreover, coliform bacteria cultured from the upper respiratory tract have been shown to be more toxic or pathogenic than coliform organisms cultured from the feces.[12] We have treated many patients who seemed to improve more rapidly when desensitizing autogenous coliform vaccine therapy was used than with other forms of treatment, although these organisms have been present in the upper respiratory tract for years in spite of all therapy.[14]

Case 5: Mr. F.B.G., aged 51, had endophthalmitis and recurrent conjunctivitis. In June 1936, Aerobacter aerogenes from the nasopharynx produced +++iritis in rabbits (very toxic for rabbits). Desensitization was begun with an autogenous vaccine. Six weeks later Aerobacter aerogenes from the naropharynx caused +++ iritis in rabbits. In January 1937 Aerobacter aerogenes from the naropharynx produced ++ iritis in rabbits (not so toxic as formerly). Two years later, Aerobacter aerogenes from the nasopharynx caused + iritis in rabbits. A new vaccine was prepared. In August 1939, Escherichia coli from the nasopharynx produced + iritis in rabbits. In January 1940 no coliform bacteria were present in the nose or throat. Again in March and January that year no coliform bacteria could be recovered. The results were ocular and general improvement.

We are indebted to Major John Bickerton for the following report of a patient treated for uveitis who seemed to respond to an autogenous vaccine.

Case 6: Sgt. R.B., white, aged 31, was under treatment at Letterman General Hospital for bilateral anterior uveitis. It was found that he had prostatic hypertrophy and vesicle neck obstruction and was operated upon for these conditions. Treatment by means of sulfonamides, intravenous typhoid vaccine, penicillin, streptomycin and fever cabinet therapy produced no improvement in the uveitis. Autogenous vaccines were prepared from prostatic secretions from which Escherichia coli, staphylococci and beta hemolytic streptococci had been isolated. Intradermal tests showed marked sensitivity to the vaccines and administration of the vaccines was associated with prompt clearing of both eyes. The routine hospital visits for urethral dilatage and prostatic massage were followed by a marked injection of the eyes and a gross increase in the number of cells in the aqueous. Therefore, a second vaccine, containing only beta hemolytic streptococcus, was prepared.

Subsequent to return to duty, the soldier neglected to take his vaccine and, on two occasions, suffered a recurrence of his uveitis. Both attacks were successfully treated by resuming the use of the second vaccine.

That ocular and cutaneous hypersensitivity are not always parallel is a fact repeatedly demonstrated in ocular tuberculosis where skin sensitivity may be low or reaction may be absent. However, if skin sensitivity is high, the eye is almost invariably hypersensitive. The difficulty in differential diagnosis is increased by the fact that the rôle of viruses and, possibly, fungi in uveal inflammations requires elucidation. In the evaluation of both positive and negative skin tests in studying hypersensitivity to bacteria, we must use all possible corollary evidence and ascertain that the specific allergic reaction is greater than that ordinarily observed in the average person of the same age group. Finally, control injections should be used to demonstrate that the patient is not merely allergic to numerous bacteria.

In testing with uveal pigment, it is well to examine the skin histologically, as suggested by Friedenwald,[40] because one of us (CB) has shown that a false positive reaction may be caused by a mild inflammatory reaction, nonallergic in nature.[4]

Diagnosis of allergy to foods is complicated by the fact that there is a masking effect, produced by the ingestion of a specific food at the time when the individual is still in a reactive phase following the last ingestion of the same food.[78] Furthermore, patients are rarely able to detect the presence of a high degree of sensitivity to wheat, eggs, milk, sugar and other foods, which are ingested by the average individual at least once a day. Skin tests for food allergies, long held suspect by us, have been proved practically worthless by the statistical studies of Rinkel.[77] We have been gratified by the results following the treatment of patients for food allergy, detected by the pulse-diet method of Coca.[28] When using the Coca technic, the pulse rate is studied before and after eating and marked tachycardia correlated with suspected foods. The elimination diets of the type suggested by Rowe[81] often serve a useful purpose.

The individual food test, as developed by Rinkel,[77] is often valuable and consists in avoiding a suspected food for four days prior to its ingestion under careful observation. A second feeding is given one hour after the first, provided no acute symptoms have developed. If the severity of coughing, sneezing or other symptoms increases markedly, the test is considered positive. If leukopenia (total leukocyte count, counting double the number of cells usually counted) of 10 per cent develops postprandially, the food is highly suspect. The count should be made before and at 20, 40 and 60 minute intervals following the initial feeding.

V. CERTAIN CONSIDERATIONS OF THE TREATMENT OF ALLERGY OF THE RETINA AND UVEAL TRACT

It has long been our contention that to neglect immunologic considerations in treating uveitis is as fallacious as neglecting rest and other general measures in treating tuberculosis. Naturally, whenever it is feasible, organisms should be eliminated. If this is impossible or inadvisable, immunotherapy should be used to desensitize or immunize the patient and all possible measures should be instituted to improve physical and mental health.

Mental and physical rest, or sometimes merely a change of mental and physical activity, will often mean the difference between success or failure in treating uveitis. This is particularly true of upper respiratory infections and tuberculosis, in which climate and sunshine must be considered. To fail to correct hypothyroidism by the use of thyroid preparations or to treat anemia or leukopenia may postpone recovery indefinitely. That other endocrine influences may be important in treating allergic manifestations of uveal inflam-

mation is indicated by the increasing attention being given to pituitary and adrenal dysfunction. This is illustrated by the work of Hench and others[53] in treating rheumatoid arthritis (considered by some as an allergic manifestation) and acute rheumatic fever with corticosteroid hormones. Although it is too early for the final evaluation of cortisone and ACTH in uveal inflammations, it seems that the more acute the process and the closer its relation to allergy, the more effective are these preparations. Chronic cases, however, are rarely benefited.[9] The action of these preparations in chronic uveitis does not seem to correspond with their apparent beneficial effect in rheumatoid arthritis, which is also a chronic disease. If tuberculosis is present or suspected, cortisone and ACTH are contraindicated.

Eradication of Generalized And Localized Infection

It is often impossible to eradicate generalized infection caused by tubercle bacilli, spirochetes, gonococci, the brucella group of bacilli and the undetermined organism causing sarcoidosis. It is also difficult, if not impossible, to remove all foci of infection harboring similar bacteria. However, every effort should be made to establish drainage, and surgery may be required to manage frank infection in the teeth and tonsils and sometimes in the sinuses. One must keep constantly in mind that even when the elimination of a focus is followed by a cure, it does not necessarily mean that the etiologic diagnosis was correct. If the threat to vision is sufficiently serious, one is often justified in proceeding radically, but it is well to remember that a focus in the upper respiratory tract (i.e., sinuses, gums and even nasal membranes) may be accompanied by a secondary infection of the gastrointestinal tract (i.e., gastritis, ileitis, cholecystitis, diverticulitis, appendicitis and possibly colitis) and that the same organisms may often be recovered from the nasal membranes and the colon.[95] Before considering radical surgery, therefore, a most careful study of the entire system should be made and other chronic infections, such as tuberculosis, sarcoidosis, syphilis and brucellosis, should be ruled out so far as possible. Furthermore, every effort should be made to raise general resistance before removing foci. Appropriate antibiotic and/or chemotherapeutic measures should be considered and often used concomitantly with the removal of foci.

General Immunity and Uveal Inflammations

Protein shock therapy is commonly employed to stimulate immunity artificially, but good results in chronic cases are uncommon and they are not without danger to life in old, debilitated and hypersensitive individuals. We place greatest reliance on typhoid H antigen or triple typhoid intravenously followed by aspiration or boiled milk administered intramuscularly. Because one may merely add to the strain on the system when using protein shock therapy, we often administer small doses of autogenous blood or other mild protein preparations intramuscularly every few days. For years we have advocated the use of autogenous vaccines because of the risks involved in the removal of foci of infection, in administering antibiotics in hypersensitive individuals and the administration of protein shock therapy.

Antibiotic or Chemotherapeutic Agents

Unfortunately, the new agents which are so valuable in combating acute infections are much less efficacious in treating chronic infections, especially of the uveal tract and retina, in which the tissues are sensitized to bacterial products and immunity is low.

After antibiotics are used, even in massive doses, resistant strains are usually left which seem to be able to per-

petuate the ocular inflammation. In the doses ordinarily employed, these products usually are merely bacteriostatic and not bacteriocidal.

Vaccines in Attempted Desensitization

In regard to the use of bacterial products in attempted desensitization, we concluded in 1936,[4] from clinical experience in treating allergic eye diseases complicated by focal infections, that it seemed to be rational therapy to eliminate infection whenever advisable and to raise resistance to these infections by vaccines, in spite of a lack of definite knowledge concerning the exact manner in which bacteria sensitize the patient.

It is still practically impossible to be certain that a certain organism recovered from a focus of infection is the actual cause of an eye lesion.[6] We have placed some reliance on intradermal tests in selecting organisms with the method proposed by Thomas, Famulener and Touart.[92] In 1929, one of us (CB)[11] stated, "Minute intradermal doses of an autogenous vaccine producing only slight local reaction are administered for several months. Their action may be entirely nonspecific, the main benefit being possibly a temporary desensitization of the patient, as the condition frequently flares up after the vaccines are discontinued."

We still use chemically killed, autogenous vaccines in chronic and recurrent uveal lesions in spite of our continued uncertainty concerning the specificity of this method of therapy. Unfortunately, we still cannot be too accurate in our etiologic diagnosis of uveal inflammations.[8]

Tuberculin

Although desensitization of the eye with tuberculin is still a subject of controversy, it seems to have a valuable place in the treatment of recurrent uveal inflammations. Wolffersdorff[98] states that, according to the greatest number of German authors, chronic recurrent uveitis is of tuberculo-allergic origin in 70 per cent of the cases. He believes that a positive intracutaneous tuberculin reaction indicates the presence, somewhere in the body, of a focus of tuberculous infection against which antibodies have been formed. The focus usually lies in the lymph nodes, and the lymphocytosis which occurs at the height of the tuberculin reaction is the result of an inflammatory process in the lymphatic system. The leukocyte count allows one to estimate the state of tissue resistance in connection with the tuberculin reaction. In case of tuberculin hypersensitiveness, there is a sudden increase in neutrophilic leukocytes, particularly in young forms, and eosinophilia. According to Wolffersdorff, tuberculin therapy is contraindicated in these cases. The author states that cases suitable for tuberculin therapy are those in which the neutrophil count remains normal or falls and the lymphocytes increase. So far no preparation has been found which is certain to desensitize the eye and avoid the possibility of producing a serious flare-up of the ocular lesion. For this reason, tuberculin therapy is particularly dangerous in paramacular chorioretinitis because of the possibility of producing an acute macular lesion which may affect both eyes. That normal eyes may also be affected is suggested by the patient tested by Weizenblatt.[97]

Case 7: A healthy young white male, with previously normal eyes, developed a bilateral neuroretinitis and cyclitis five days after a routine tuberculin test, made by Weizenblatt. The skin reaction was ++++. Eventually complete recovery occurred. Six and one-half years later, another tuberculin test was followed seven days later by a bilateral neuroretinitis and cyclitis. Complete recovery followed. There was freedom from ocular symptoms between the tuberculin tests.

These ocular complications following injection of tuberculoprotein may be

termed a delayed allergic reaction, such as is sometimes observed in serum diseases.

The use of tebeprotein for diagnosis and treatment is favored by Urbanek[93] and Mohamed[68] because it contains specific tuberculous protein.

VI. SUMMARY AND CONCLUSIONS

Certain manifestations, considered allergic in nature, in lesions of the retina and uveal tract may explain some of the clinical signs and diseases in which the etiology is highly controversial. Lesions in the uveal tract, when frankly granulomatous, are believed by some to be an expression of allergy to living bacteria in the lesions. The nongranulomatous uveal diseases may be an expression of allergy to bacterial products alone, although the presence of bacteria even in these lesions cannot be definitely excluded. Various etiologic factors have been implicated in uveitis. The one which is most controversial is focal infection, and especially the allergic theory of the causation of uveitis. Our animal experiments and clinical experience with attempted desensitization of patients with bacterial products has strengthened our belief in the allergic theory of the etiology of some of these uveal and retinal lesions.

It is also highly debatable whether syphilis produces retinal and uveal lesions through an allergic reaction. Inability to determine skin hypersensitivity in these cases makes the diagnosis of allergy difficult. However, the pathologic picture found in certain uveal lesions is considered allergic when observed in other parts of the body. Furthermore, similar pathologic findings are considered as caused by allergy when other bacteria are implicated.

Uveal tuberculosis has received more intensive study than syphilis and many consider the lesions as manifestations of hypersensitivity to the tubercle bacilli. Furthermore, for many years, ophthalmologists have used preparations of tubercle bacilli in minute doses in an attempt to desensitize their patients and this was done before allergy was generally recognized as a factor in producing ocular pathology.

Although sensitization to uveal pigment may be a factor in sympathetic ophthalmitis, the experiences reported here lead us to the conclusion that bacterial allergy should be considered. The lessened incidence of sympathetic ophthalmitis following the use of antibiotics or treatment of infected foci is possibly a point in favor of this concept.

That hypertension in the central artery of the retina may be associated with allergy is strongly suggested by clinical findings in allergic patients and by animal experiments.

Moreover, from the clinical and experimental data reported here, it is believed that retinal separation may be caused by allergy in certain individuals.

Since angioneurotic edema is considered to be an allergic manifestation, it seems reasonable to believe, from some of the evidence presented, that an angioneurotic allergic type of swelling could explain certain angiospastic retinopathies, especially in patients with vascular instability.

Dermatologists long have favored the bacterial allergy theory of the causation of lupus erythematosus. That the retinitis associated with this disease may be similarly caused is certainly worthy of consideration.

It is possible that acute exudative macular chorioretinitis can be explained on the basis of allergy because of the characteristic pathological condition and the beneficial effects of vasodilation.

Moreover, it is contended that glaucoma secondary to uveitis is often a manifestation of allergy and that in all

likelihood, allergy can explain certain cases of acute primary glaucoma. Some evidence which seems to support this contention is presented and discussed. That chronic primary glaucoma may be related to allergy was previously suggested. It is believed that exacerbations of tension in association with increased activity in chronic sinusitis may be the expression of a mild allergic edema in the uveal tract. Our experience indicates that tension is sometimes more easily controlled or visual fields improved when allergies to food are eliminated by proper diet and when foci of chronic infection are drained or completely eliminated, or the patients are desensitized with autogenous vaccines.

In diagnosing allergy of the uveal tract and retina, dermal and intradermal tests for hypersensitivity have to be relied upon heavily. However, even in tuberculosis, ocular and cutaneous hypersensitivity are not always parallel. If skin hypersensitivity to tuberculin is marked, the eyes are almost invariably hypersensitive. In evaluating skin tests, we must be certain that the specific allergic reaction is greater than that ordinarily observed in the average person of the same age. All possible corollary evidence must be evaluated. For example, in assessing the importance of an intradermal reaction to uveal pigment, histologic studies should be made to exclude false positives.

In treating allergic lesions of the uveal tract and retina, all measures to improve the general condition and eliminate nervous tension should be combined with attempted desensitization to known or suspected allergens, when they cannot be eliminated. Some benefit seems to result from desensitization with tuberculin in suspected tuberculous uveitis and retinal hemorrhages in adolescents, if the doses are minute and reactions, either local, general or focal, can be avoided.

Desensitization with uveal pigment seems to be useful in the treatment of sympathetic ophthalmitis but bacterial desensitization should also be considered.

In allergy associated with focal infection, best results are obtained when the focus can be completely eliminated or well drained, as in some patients with chronic sinusitis. Desensitization with minute doses of chemically killed autogenous or stock vaccines seems to be beneficial when lesions recur and the suspected bacteria persist in spite of surgery and the use of antibiotics, cortisone and sulfonamides.

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Symposium: Ocular Allergy

CORNEA AND LENS

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NEW YORK, N. Y.

I AM greatly honored to appear before this Society to discuss the subject "Manifestations of Ocular Allergy on the Cornea and Lens." Having selected me from among many that could have discussed this problem with great authority, would seem to imply that I am an expert on this subject. Nothing could be further from reality. I am just an ophthalmologist that has become interested in this subject observing cases of ocular inflammation that could not be explained by anything else but allergic reactions. My only rôle before this Society is to present a few clinical facts, trying to interpret them afterwards with my limited knowledge of the problem.

CORNEA

Allergy has been ascribed as the cause of corneal edema, vesicle formation, herpes of the cornea, herpes zoster ophthalmicus, some types of ulcers, etc. I first became interested in the subject of ocular allergy after observing the postoperative complications which took place in patients that had undergone corneal surgery. Patients on whom corneal transplantations were performed with uncomplicated recovery, discharged from five to eight weeks after the operation with a clear graft and greatly improved vision, on several occasions returned within a few days, weeks, months or years complaining of sudden diminution of vision, coincident with the development of a severe head cold or sore throat.

Case Reports

1. J.G. Diagnosis: Advanced keratoconus, right eye. Vision: Fingers at 2 feet. Keratoplasty, left eye, partial penetrating square, 6.25 mm. Uneventful recovery. Discharged six weeks later with transplant clear; vision, with correction, 20/25. Six weeks later severe sore throat, developing uveitis immediately. Edematous graft (fig. 1A) and numerous keratic precipitates in posterior surface of the graft. Vision, 20/200. Streptococcic tonsillitis found. Tonsils removed, atropine instillations locally and intravenous typhoid injections and penicillin systemically. The eye quieted down shortly after removal of the tonsils. Transplant cleared rapidly, and seven weeks later transplant was clear; patient discharged with vision 20/20-2 (fig. 1B).

2. A.S. Diagnosis: Corneal opacity in pupillary area, left eye, resulting from ulcerative keratitis. Vision, 20/300. Keratoplasty, partial penetrating square, 7 mm., with uneventful recovery. Two months later transplant was clear; vision, 20/50. One month afterwards the patient developed severe head cold followed by uveitis, rendering the graft cloudy (fig. 1C). Vision, fingers at 1 foot. Treated locally with atropine instillations and systemically with intravenous typhoid injections and antibiotics. Ocular inflammation subsided within three weeks. Graft cleared slowly. One year later vision with correction, 20/50+3. Two years later vision, 20/25-3 (fig. 1D). (fig. 1D).

3. A.S. Diagnosis: Advanced keratoconus, left eye. Vision, 10/400. Keratoplasty, partial penetrating square, 6.5 mm. Uneventful recovery until four weeks postoperatively when uveitis developed, rendering the graft cloudy (fig. 1E). Treated locally with atropine instillations and systemically with intravenous typhoid injections and antibiotics.

From Columbia University College of Physicians and Surgeons.
Presented at the meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, Oct. 19, 1951, Chicago, Ill.

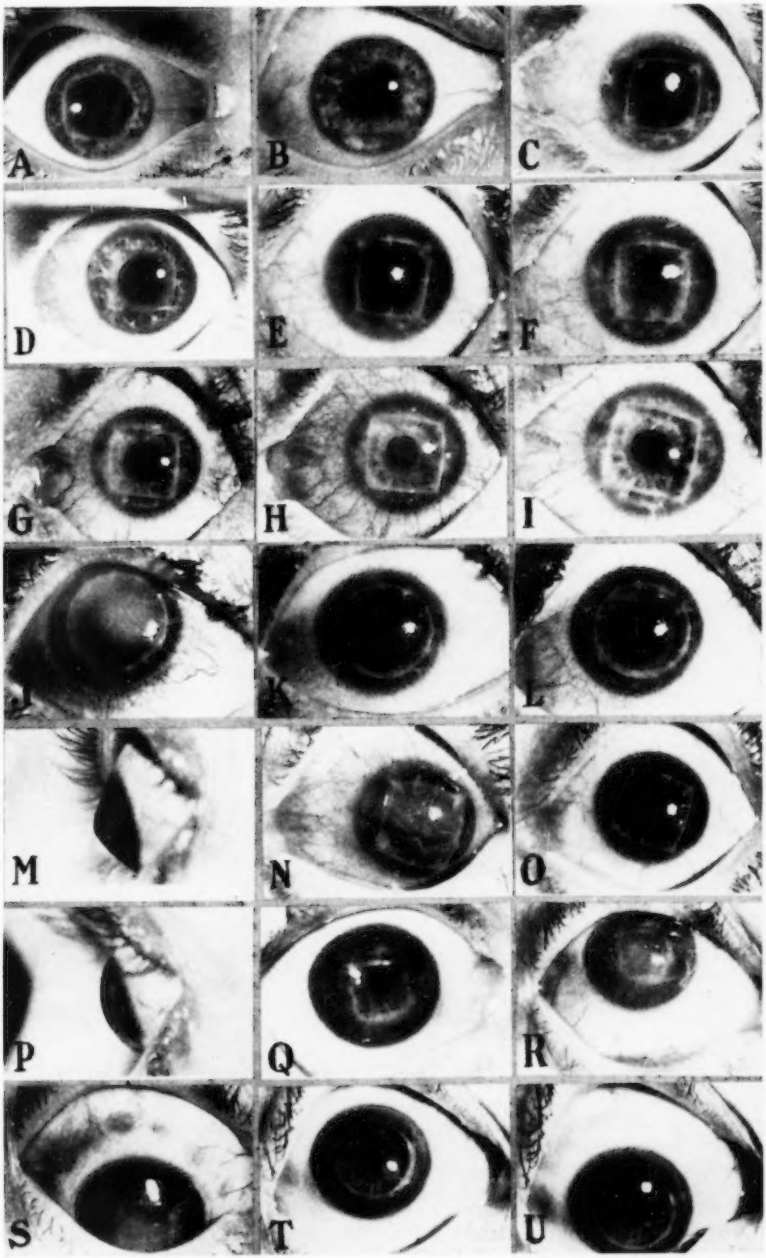

FIG. 1—Photographs illustrating cases from 1 to 7.

The eye failed to improve. Pathologic nasal condition found, which was treated surgically, resulting in the immediate improvement of the ocular inflammation and complete recovery in a short time. The transplant cleared gradually without treatment. Vision one year later, 20/40-2 (fig. 1F).

4. R.H. Diagnosis: Advanced keratoconus, left eye. Vision: fingers at 1 foot. Keratoplasty, partial penetrating square, 7 mm., with uneventful recovery. Discharged two months later. Vision, 20/20 with correction (fig. 1G). One month later, following very severe head cold, patient returned complaining of very cloudy vision. Eye inflamed and painful, was affected with uveitis, pronounced perikeratic injection, transplant hazy (fig. 1H). Vision, 20/60. Treated locally with atropine, 1 per cent, and cortisone instillations, and systemically with intravenous typhoid injections and antibiotics. Inflammatory symptoms subsided within two weeks. Transplant cleared completely and patient was discharged with vision of 20/20-1 (fig. 1I).

5. S.G. Diagnosis: Advanced keratoconus, right eye. Vision, fingers at 10 feet. Keratoplasty, partial penetrating round, 7.5 mm. Uneventful recovery. Discharged one month later with clear transplant. One week later, following attack of head cold with involvement of ethmoids and sinuses, developed uveitis. The transplant became cloudy (fig. 1J). Cortisone instillations and cortisone orally failed to stop the inflammation or clear the transplant. Patient hospitalized, treated by intravenous typhoid injections and antibiotics systemically and with atropine instillations locally. Within two weeks the inflammation subsided and the transplant cleared considerably (fig. 1K). Three months later transplant fairly clear. Vision, 20/40 (fig. 1L).

6. O.L. Diagnosis: Advanced keratoconus, left eye (fig. 1M). Vision, fingers at 1 foot. Keratoplasty, partial penetrating square, 7.5 mm. Uneventful recovery for the first five weeks. Suddenly developed uveitis and the transplant became very cloudy (fig. 1N). Atropine and cortisone instillations locally and cortisone systemically failed to improve the eye. The patient was found to be affected with deflected septum and left ethmoiditis and sphenoiditis. Surgical treatment of the pathologic nasal condition immediately cleared ocular inflammation. Two months later the eye was quiet, transplant still somewhat hazy and with slight edema but clearing rapidly. Patient was discharged for treatment elsewhere. Vision, fingers at 3 feet (fig. 1,O and P). Three months later patient reports he is able to read with operated eye without glasses.

7. K.C. Two years previously the patient had developed corneal opacity in pupillary area, resulting from ulcerative keratitis. Vision, fingers at 4 feet. Keratoplasty, partial penetrating square, 5 mm. Discharged six weeks later with vision 20/200 (fig. 1Q). Twelve years later, following acute attack of left ethmoiditis, the eye developed uveitis and secondary glaucoma. Tension, 65 plus. Transplant became cloudy, and vision diminished to fingers at 2 feet (fig. 1R). The ethmoiditis was treated surgically. The inflammatory reaction of the eye subsided in a few days, but tension remained high in spite of myotics. Cyclodiathermy performed; eight perforating electrocoagulations, 6 mm. from the limbus around the upper half of the globe, reduced the tension within normal limits (fig. 1S). One year later, the transplant still remained cloudy. Vision, fingers at 12 inches. Tension, 23. Keratoplasty, partial penetrating, round, 6.5 mm., performed with uneventful recovery. Patient discharged five weeks later. One year later, transplant still clear. Tension, 23. Vision, 20/200 (fig. 1, T and U).

These are typical cases, of which I could report several dozen. The realization that acute pathology of the nose and throat seemed to play an important rôle in the postoperative complications of patients who had undergone corneal transplantation focused my attention on the possibility of other corneal affections that could be related to acute or chronic diseases of the upper respiratory tract. The following cases seemed to be typical examples of ocular complications arising from diseases of the nose and throat on the basis of bacterial allergy.

Case Reports

8. B.D. Child, 7 years of age, affected with recurrent attacks of keratoconjunctivitis for five months. Right eye, with pronounced photophobia, blepharospasm and lacrimation, showed extensive superficial corneal opacity. Child found to be affected with adenoids and hypertrophic tonsils. Vision, light perception and projection. Very uncooperative on account of photophobia and blepharospasm. One month after removal of infected tonsils and adenoids, the inflammatory symptoms of the

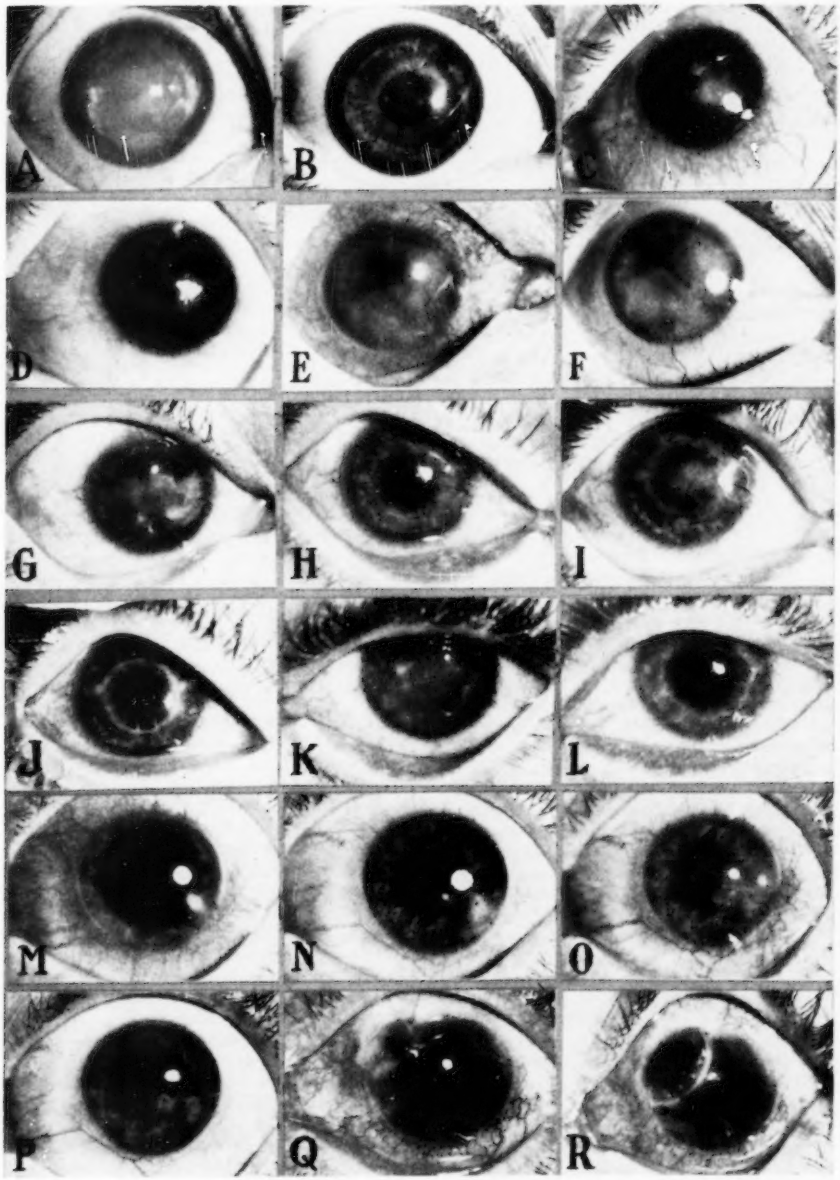

FIG. 2—Photographs illustrating cases from 8 to 13.

eye subsided (fig. 2A). Corneal opacity remained. Seven months later partial lamellar keratoplasty, 9 mm., performed with uneventful recovery. Vision, one year later, fingers at 10 feet (fig. 2B).

9. G.S. Patient treated elsewhere for severe keratitis which failed to improve by local treatment with atropine, cortisone instillations, cortisone subconjunctivally and antibiotics systemically. General check-up resulted in negative findings. Eye showed perikeratic injection and corneal opacity in pupillary area surrounded by interstitial corneal infiltration (fig. 2C). Vision, 20/400. Otorhinolaryngologic examination revealed deflected septum and pansinusitis with pronounced involvement of the ethmoids and antrums. Treated locally with atropine instillations and surgical treatment of nasal pathology. Automatically, the ocular inflammatory symptoms subsided after nasal intervention. Six weeks later the eye was quiet, the corneal infiltration disappeared. Cornea considerably clearer and vision improved to 20/40-2 (fig. 2D).

10. L.G. Right eye inflamed for six months. Treated elsewhere with diagnosis of ocular tuberculosis. Failed to improve by local or general treatment including tuberculin. Found to be affected with pronounced perikeratic injection, corneal opacity extending over the whole cornea with superficial vascularization (fig. 2E). Vision, fingers at 6 inches. General examination failed to reveal any evidence of tuberculosis or any other cause of the ocular inflammation with the exception of deviated septum with bilateral ethmoidal and maxillary sinusitis. These conditions were surgically treated without any additional treatment. The ocular inflammation subsided, corneal vascularization receded, but the dense corneal opacity remained. Six months later the eye is still quiet. Vision, fingers at 2 feet (fig. 2F). The corneal opacity will require treatment by surgery.

11. P.B. Recurrent attacks of keratoconjunctivitis for 15 years. Diagnosis of ocular tuberculosis made elsewhere. Unsuccessfully treated with tuberculin. Both eyes affected with pronounced photophobia, blepharospasm and lacrimation, perikeratic injection and superficial corneal opacity in pupillary area. Vision, right eye, fingers at 2 feet; left eye, fingers at 5 feet. Photographs could not be taken because of the intense photophobia, blepharospasm and lacrimation. General check-up failed to reveal evidence of tuberculosis. Instead, severe pansinusitis with deviated septum discovered. Pathologic nasal condition treated surgically, requiring extra and intranasal intervention. The inflammatory ocular symptoms subsided shortly afterwards making it possible to photograph the eyes (fig. 2 G, right eye; fig. 2K, left eye). Partial lamellar keratoplasty of 6 mm. in the right eye and 7 mm. in the left eye was performed with uneventful recovery. Patient discharged one month later. Vision, both eyes 20/200 (fig. 2H, right eye; fig. 2L, left eye). Six months later vision in the right eye failed to 20/400, the transplant becoming cloudy (fig. 2I). Re-infection of sinuses was found and treated conservatively. Two weeks afterwards, keratoplasty, partial penetrating, round, 6 mm., was performed with uneventful recovery. Two months later, transplant slightly hazy. Vision, 20/200 (fig. 2J).

12. J.H. Recurrent attacks of corneal abscesses (fig. 2, M, N and O) for one and a half years failed to clear with antibiotics locally and systemically, necessitating during each attack, curettage and electrodessication. Sterile pus recovered from the abscesses. General check-up resulted in negative findings with exception of sinusitis. This condition was treated surgically one and a half years after the onset of the keratopathy, with the immediate disappearance of the ocular inflammation, which has not recurred since the nasal pathology was adequately treated. Vision, which was 20/25 in the first examination, reduced to 20/40 (fig. 2P).

13. A.P. Patient treated elsewhere for keratitis with corneal vascularization in upper nasal quadrant. A lamellar keratectomy followed by radiation resulted in partial staphyloma of the cornea with a large descemetocele at the limbus (fig. 2Q). The patient was referred to me for keratoplasty in order to reinforce the weakened portion of the cornea and to prevent perforation. The patient was found to be affected with pronounced blepharospasm, photophobia and lacrimation in both eyes. Both eyes showed pronounced conjunctival injection. The left eye had a partial corneoscleral staphyloma in the upper nasal quadrant. Vision, fingers at 1 foot. A pathologic nasal condition with which the patient was affected was treated surgically, upon which the ocular inflammatory symptoms subsided. Five months later, the staphyloma was excised and the defect closed by partial penetrating keratoplasty, 7 by 5 mm. Transplant remained fairly clear. Eight months later, transplant still clear, pronounced astigmatism (fig. 2R). Vision, fingers at 1 foot. Keratoplasty in the center of the cornea will be required for visual purposes.

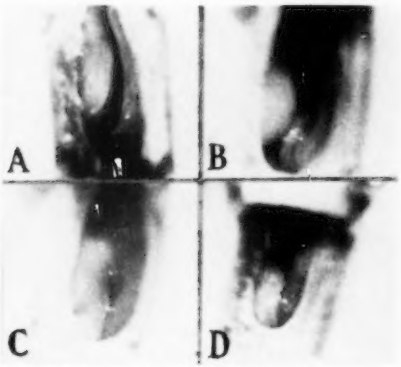

FIG. 3—Photographs illustrating appearance of the nose with various degrees of nasal pathology casually observed by the ophthalmologist.

So far, in the cases reported, the relation between cause and effect was so obvious that no doubt was entertained regarding the causative agent of the ocular complications. However, there were other cases of primary corneal affections or complications after corneal surgery in which thorough general check-up failed to reveal any possible cause for the disease of the eye. All these cases, however, frequently showed a common denominator: the presence of more or less pronounced findings in the nose which might explain, at least in part, the inflammatory symptoms of the eye. Nasal objective findings consisted generally of a deviated or thickened septum, congested nasal mucosa and hypertrophic turbinates. These findings were grossly detected by a casual observation of the nose through the nostrils after completion of the ophthalmologic examination (fig. 3). Occasionally the tonsils also showed a pathologic appearance. Subjectively, the patients were sometimes conscious of nasal discomfort, such as postnasal drip, frequent colds, headaches, pain around the eyes and accommodative difficulties. Otorhinolaryngologic reports in these cases were usually negative, including transillumination, roentgenograms and other clinical examinations. The nasal findings observed by the ophthalmologist were often attributed by the otorhinolaryngologist to allergy and, at any rate, there was not sufficient nasal pathology to explain the ocular complication. These cases, subjected to conservative measures such as antibiotics, protein therapy, anti-allergic medication, general supportive measures and, more recently, cortisone, failed to be cured or even improved. The minor nasal symptomatology was then suspected to hide more pronounced infection and the patients were submitted to corrective nasal surgery. In all these cases, surgery revealed sinus involvement, sometimes moderate, sometimes advanced, even to the point of bone necrosis. All cases were either greatly improved or completely cured after surgical intervention, proving beyond doubt that the factor of the ocular complication lay in the nasal infection.

Case Reports

14. E.L. Diagnosis: advanced keratoconus, left eye. Vision, fingers at 8 feet. Keratoplasty, partial penetrating, square, 6.5 mm., performed. Uneventful recovery for three weeks, then uveitis and gradual cloudiness of the graft took place. General check-up, negative. Treated locally with atropine and hot applications; systemically with antibiotics and intravenous injections of typhoid. Ocular inflammation subsided leaving the transplant cloudy. Vision two years later, 20/400. Second partial penetrating keratoplasty, 5.5 mm., round, within the previous graft performed. Recovery uneventful for three weeks, then uveitis developed. Before transplant began to get cloudy, patient was hospitalized, treated locally by atropine instillations and hot applications and systemically with intravenous typhoid injections and antibiotics. Inflammation subsided within a few weeks, transplant remaining clear. Two months later, vision 20/40; one year later, transplant clear. Vision with correction, 20/20 and Jaeger No. 1 (fig. 4A.) Right eye, advanced keratoconus. Vision 20/400. Keratoplasty, partial penetrating, 6.25 mm., square, performed. Uneventful recovery for two weeks, then uveitis developed. Local and systemic treatment with antibiotics and typhoid injections failed to prevent opacification

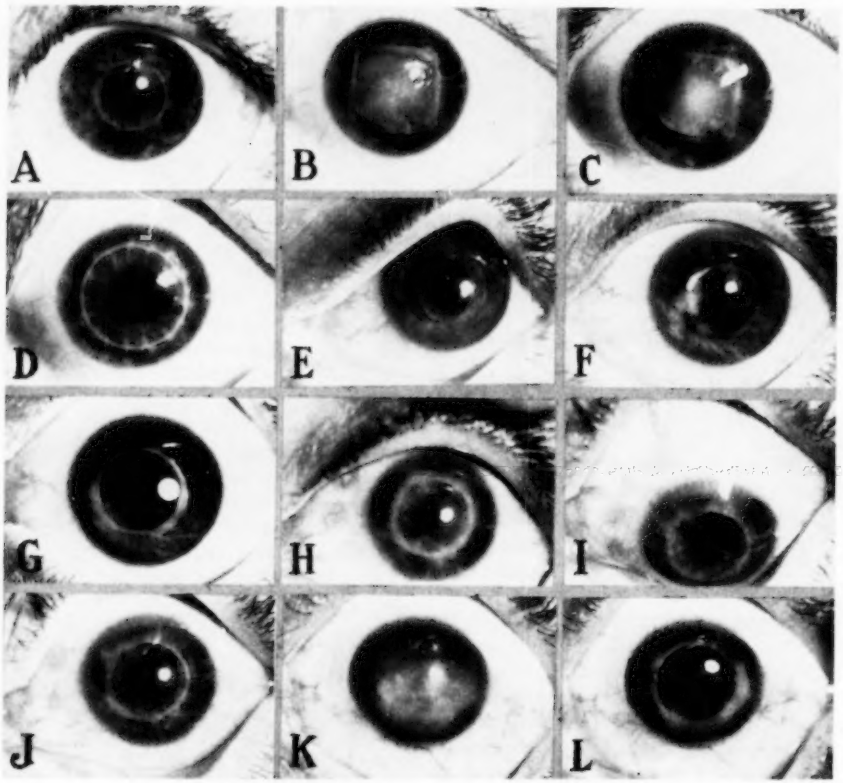

FIG. 4—Photographs illustrating cases from 14 to 17.

and edema of the graft (fig. 4B). General check-up revealed only chronic sinusitis, which was treated conservatively without improving cloudiness of the graft. Six months later a partial lamellar keratoplasty, 6 mm., was performed. Corneal edema invaded the transplant, rendering it opaque (fig. 4C). Vision, fingers at 1 foot. The patient developed rheumatoid arthritis, which was systemically treated with cortisone. Twenty months later, while under systemic treatment with cortisone, a third partial penetrating keratoplasty, 7 mm. round, was performed. Recovery uneventful for three weeks, then mild uveitis developed with slight cloudiness of the graft. In spite of the cortisone systemically, which the patient continued to use during recovery, no delay in healing was observed. The eye was treated locally with cortisone instillations. The ocular inflammation subsided and the transplant cleared within two weeks with slight edema persisting. Six months later, transplant still slightly edematous but quite clear. Vision, 20/200 (fig. 4D).

15. G.E. Diagnosis, left eye, corneal opacity in pupillary area following keratitis after removal of embedded foreign body two years previously. Vision, 20/300. Keratoplasty, partial lamellar, 6 mm., with uneventful recovery. Discharged one month later with vision 20/400. Six months later, vision 20/50+2 (fig. 4E). Patient affected with chronic sinusitis. During the following year the eye was always somewhat congested, and the patient had several attacks of kerato-iritis. The transplant developed fatty degeneration, which increased slowly. Vision diminished to 20/200 (fig. 4F). The chronic sinusitis, which had been conservatively treated without improvement of the eye, was finally treated by surgery. With this treatment, the inflammatory ocular symptoms subsided. Partial penetrating kerato-

plasty was performed with uneventful recovery. One year later the patient was without any sign of ocular inflammation. Transplant clear. Vision with correction, 20/20 and Jaeger No. 1 (fig. 4G).

16. F.G. Diagnosis: Advanced keratoconus, both eyes. Vision, right eye, 2/200; left eye, 5/200. Right eye, keratoplasty, partial penetrating, square 4 mm. Uneventful recovery. Thirteen years later transplant still clear. Vision with correction, 20/20+2 for distance and Jaeger No. 1 for near.

Left eye, keratoplasty, partial penetrating, square 6 mm. Uneventful recovery for three weeks, then uveitis and cloudiness of the graft developed. Not improved by local or systemic treatment. Vision one year later, fingers at 2 feet. One year later, re-transplant, partial penetrating, round 6.25 mm., with the same course as in the previous transplant, graft remaining hazy. Three years later the transplant cloudy, vision 20/200. Three years later, third partial penetrating keratoplasty, 6.5 mm. round. Uneventful recovery for three weeks, then uveitis developed. General check-up negative with the exception of chronic sinusitis. The eye failed to improve by local or systemic treatment. The nasal condition was treated by surgery, upon which ocular inflammation subsided. The eye developed secondary glaucoma. Tension is reduced to within normal limits by cyclodiathermy, using a 1.5 mm. electrode, eight penetrating electropunctures 7 mm. from the limbus around the upper half of the eye. Two months later, tension is normal; transplant cloudy; vision, fingers at 2 feet (fig. 4H). Transparent cleared gradually and two years later transplant was clear, vision 20/20-2, and tension normal (fig. 4, I and J).

17. J.S. Diagnosis: Left eye, keratitis with corneal opacification for five months, unimproved by local and systemic treatment. Vision, fingers at 4 feet (fig. 4K). General check-up negative. Otolaryngologic reports conflicting; one reporting negative findings, another sinusitis on the side of the affected eye, mostly in the ethmoidal and sphenoidal region. Surgical treatment of nasal condition resulted in subsidence of ocular inflammation. Five months later keratoplasty, partial lamellar round, 6 mm. Uneventful recovery. Four months later, vision 20/50-1. Three months later the eye became inflamed and vision failed to 20/300 (fig. 4L). Otolaryngologist's report negative. Eye continued to be inflamed and the transplant slowly and gradually became cloudy (fig. 5A). A second keratoplasty, partial penetrating, round 6 mm., was performed seven months later. Again uveitis developed (fig. 5B). Upon the findings of the eye alone, the consulting otolaryngologist advised a sinus operation through the external approach, finding ethmoids, sphenoids and frontals on the side of the affected eye with advanced polypoid degeneration of the mucosa, pus and involvement of the periosteum and bone in spite of the fact that roentgenograms had been found to be negative. After the nasal intervention, the ocular inflammation subsided. Transplant cloudy. Vision, fingers at 2 feet (fig. 5C). Glaucoma developed but was controlled by cyclodiathermy—eight perforating electrocoagulations with 1.5 mm. electrode, 7 mm. from the limbus in the upper half of the globe. Tension remained within normal limits, transplant gradually cleared. Seven months later tension is still within normal limits. Vision improved to 20/200 (fig. 5D). If further improvement in vision takes place, a third corneal transplant may be avoided.

18. E.B. Diagnosis: Groenouw's corneal dystrophy, both eyes. Vision, right eye 20/60; patient unable to carry on professional activities. Keratoplasty, partial penetrating, round 5 mm., performed with uneventful recovery. Two years later, vision with correction 20/20-1 and Jaeger No. 1 (fig. 5E). Two years after operation in right eye, left eye vision 20/80. Left eye keratoplasty, partial penetrating, round 6 mm. Immediate postoperative recovery uneventful. Four weeks later, uveitis with progressive cloudiness of the graft developed. Uveitis unaffected by local treatment with atropine and cortisone instillations and systemic treatment with antibiotics and intravenous typhoid injections and cortisone. Vision nine months later, fingers at 6 feet. General check-up negative with the exception of chronic sinusitis. One year later re-transplant, partial penetrating, 7 mm. round, followed by the same postoperative course. Recovery uneventful for three weeks; then uveitis (fig. 5F) with progressive opacification of the graft, developing ocular inflammation failing to respond to local and systemic treatment using cortisone, antibiotics and intravenous typhoid injections. Sinusitis treated by surgery and ocular inflammation subsided. Transplant remained cloudy for one year without tendency to clear. Vision, fingers at 2 feet (fig. 5G). A third keratoplasty will be necessary to improve vision.

19. I.K. For five years affected with recurrent keratitis in the left eye, developing a dense corneal scar covering the pupillary area (fig. 5H). In addition to the dense opacity,

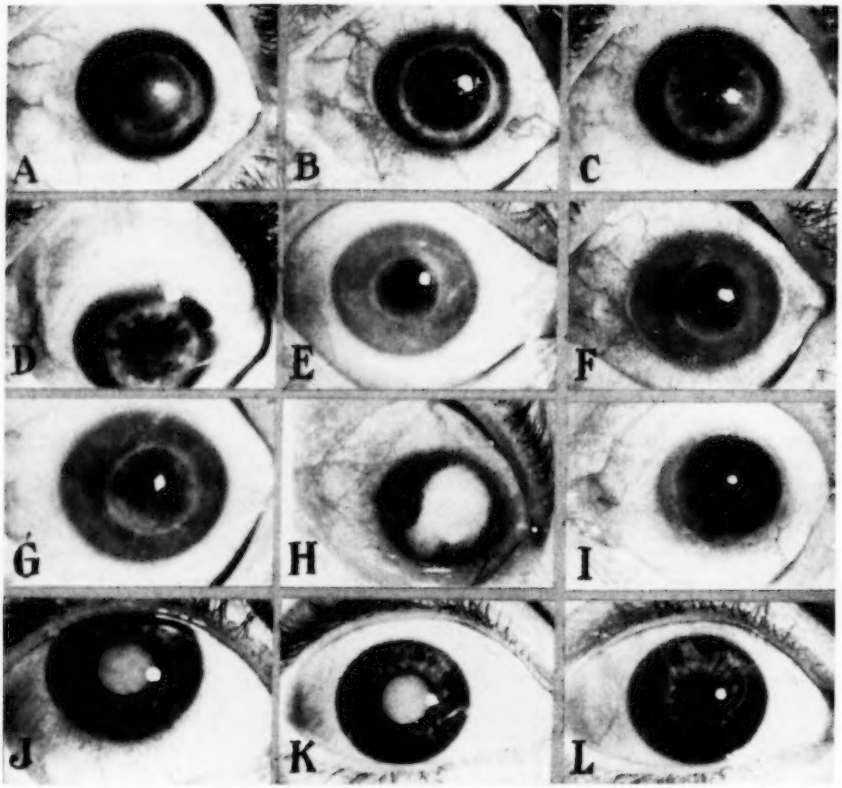

FIG. 5.—Photographs illustrating cases from 17 to 20.

the eye showed pronounced perikeratic injection with marked photophobia, blepharospasm and lacrimation. The fellow eye remained within normal limits but was also affected with marked conjunctival congestion. Vision, left eye, hand motion at 1 foot. The eye failed to improve by local or systemic treatments. General check-up was negative with the exception of advanced pansinusitis. Surgical treatment of nasal condition resulted in immediate subsidence of all ocular inflammation and discomfort, without recurrence of keratitis. A subtotal penetrating keratoplasty of 10 mm. was performed on the left eye. Uneventful recovery. Six months later, eye quiet, transplant clear, vision with correction 20/50 (fig. 51).

LENS

Regarding lens changes of an allergic nature, the direct action that lens cortex has on the uveal tract when an extracapsular cataract extraction has been performed is well known. This local sensitivity to lens protein, called phacoanaphylactic reaction, has been observed by all ophthalmic surgeons. Cataracts occurring with neurodermatitis and eczemas have also been reported, although the interrelation of the two affections has been questioned by many. The posterior subcapsular cataract, which is often observed in patients when a thorough examination fails to discover any systemic disease such as tuberculosis, syphilis, endocrine dysfunction, malignancies or dietary deficiencies, could be related to humoral

imbalance of the eye, caused by a hidden focus of infection (possibly in the sinuses) supplying the antigen for bacterial allergy.

Some postoperative iritis and uveitis of uncomplicated extractions of senile cataracts can also be traced to bacterial allergy, requiring in most cases the elimination of the bacterial antigen in order to effect the subsidence of the inflammatory reaction. The following case shows a clear-cut association of sinus infection with the development of a complicated cataract.

Case Report

20. A.M. Diagnosis: Uveitis, right eye for two months, developing a complicated cataract (fig. 5J). Pronounced photophobia, blepharospasm and lacrimation present. Severe pain in and around the eye. Moderate keratic precipitates, advanced cataractous changes in pupillary area. General check-up negative with the exception of sinus infection, which was treated surgically. Symptoms of ocular inflammation, as well as discomfort, subsided (fig. 5K). Vision: light perception and projection. Six months later, intracapsular cataract extraction with peripheral iridectomy was performed, round pupil. Uneventful recovery. For five months no recurrence of inflammation or discomfort has taken place. Vision with correction, 20/30 (fig. 5L).

Some cases of fatigue of accommodation could be explained on the basis of bacterial allergy. All ophthalmologists are acquainted with cases of young patients in the age group of the 20's or early 30's with a minor error of refraction within a half diopter of hyperopia or astigmatism, or no error of refraction at all with a perfect muscle balance, and yet they complain of inability to read for an extended period of time. Extreme discomfort in close work, tiredness of the eyes and the usual asthenopic symptoms are often associated with frequent congestion of the eyes and severe headaches. The presence of this lack of proportion between the absence of objective findings and the pronounced discomfort of the patient suggests the presence of an extraocular factor responsible for the fatigue of accommodation. In these patients, thorough check-up usually fails to reveal any possible cause for the discomfort with the exception of nasal pathology, which is found in a high percentage of cases. When such nasal conditions are neutralized by conservative means, or by the removal of the focus of infection when necessary, the discomfort usually disappears, proving the nature of the etiologic factor.

DISCUSSION

I shall attempt briefly, from the limited point of view of a clinical ophthalmologist, to give an interpretation which may be accepted, modified or entirely rejected if this interpretation cannot be substantiated by further clinical study.

For purposes of discussion, allergies are usually divided into two groups: (1) those due to inhalants and foods (protein sensitivity) and such drugs as penicillin, atropine, etc.; and (2) those due to bacterial products.

The spectacular results attained by the elimination of the offending agents in the first group are well known to everyone and need no further comment on my part. In my talk I will limit myself entirely to the latter group.

In the corneal cases reported, there seems to be no doubt that the presence of infection in the upper respiratory tract was the primary factor in the development of the corneal disease, either of the primary lesions or the changes secondary to surgery. At first, as long as the changes in the cornea are mild (such as edema and infiltration) the neutralization or removal of the responsible factor will immediately stop the ocular manifestations and the eye will be restored to its normal functioning state without damage or sequela. If the factor responsible for the ocular inflammation has remained unchecked long enough to cause more deterioration of the corneal tissues, mainly vasculariza-

tion and fibrosis, permanent damage will result, requiring further treatment to the eye besides the final elimination of the causative agent.

The allergic reaction alone seems sufficient to explain the first manifestation of corneal disease as the direct response of the cornea to the antigen. This reaction is sometimes mild, sometimes pronounced, with marked vascularization, infiltration, ulceration, and eventually scar formation. Other changes in the cornea cannot be explained by direct allergic reaction in the cornea but rather must be attributed to trophic changes brought about by the disease of the fifth nerve. Corneal edema, epithelial or endothelial vesicular formation, recurrent ulceration and opacification seem to be the result of trigeminal disease reaching the cornea through the posterior ciliary nerves, apparently the nerves most responsible for supplying corneal innervation.

In 1951, E. Zander and G. Weddell,[4] experimenting on rabbits, found that the majority of nerve fibers in the rabbit reach the cornea via the ciliary nerves. They also found that, normally, few nerve fibers reach the cornea other than by way of the fifth nerve. As a result of section or destruction of the ciliary nerves or the gasserian ganglion, they concluded that the rabbit cornea is supplied solely by the axons of the fifth cranial nerve reaching the cornea by way of the ciliary nerves.

In treating corneal disease primary or secondary to surgery, if the ocular signs of inflammation do not disappear rapidly by the neutralization of the focus or foci of infection through the administration of antibiotics, protein therapy, antihistaminics, cortisone and other general supportive measures, the focus or foci of infection should be removed without delay to prevent trophic changes caused by trigeminal disease and permit, in cases of keratoplasty, the unhindered regeneration of corneal nerves, without which permanent transparency of the graft does not seem to be possible.

The sympathetic nerve may also be involved. This involvement of the sympathetic and paralysis of the vasoconstrictors in cases of nasal pathology may be explained by the link on the ciliary ganglion of the sympathetic fibers coming from the carotid plexus and fibers from the ophthalmic branch of the trigeminal nerve. The diseased second branch of the trigeminal nerve may affect the sympathetic nervous system through the ophthalmic branch at the ganglion or, more likely, sinus disease will cause, by bacterial allergy, the affection of both the sympathetic and the trigeminal nerve, either separately or at their link in the ciliary ganglion.

The difficulties of accommodation in nasal disease could be explained by the link at the ciliary ganglion of the third nerve, the sympathetic and the ophthalmic branch of the trigeminal nerve by either the trigeminal nerve affecting both the third nerve and the sympathetic in their link at the ganglion or, more likely, sinus disease affecting by an allergic reaction these three elements, separately or together.

Several quotations will be of interest in this discussion.

In 1912, W. H. Haskins examined 600 skulls and found septal deviation in 75 per cent.

In 1949, in the proceedings of the Fourth International Congress of Otolaryngology, C. W. Bullard,[1] in a paper analyzing 2,600 cases of sinusitis in respiratory allergy in children, summarized his findings as follows:

1. Eighty per cent of otitis media cases were associated with maxillary sinus pathology, 76 per cent of which developed upper respiratory allergy.
2. Forty-six per cent of children with sinus pathology developed upper respiratory allergy later in life.

3. Chronic purulent maxillary sinus pathology was found in 78 per cent of all adult asthmatics studied.
4. Seventy-three per cent of all hay-fever patients studied showed maxillary sinus pathology, even in the quiescent stage.
5. Fifty-four per cent of cases with purulent maxillary sinus pathology developed upper respiratory allergy later in life.

His conclusions from the above findings were:

1. Asthma and hay fever are the result of progressive sinus pathology.
2. Chronic purulent maxillary sinusitis is common in the absence of sinus symptoms.
3. The history of otitis media is important in determining the past history of sinus disease.
4. Chronic bronchitis is usually secondary to a purulent maxillary sinusitis.

In 1951, Kretzschmar and Jacot[2] reported the case, of eight years duration, of a young patient, slightly over 20 years of age, who after a mild head trauma presented the following syndrome:

Objective symptoms:
1. Unilateral injection of the conjunctiva without secretion and with intermittent epiphora
2. Recurrent edema of the lids of the right eye with false ptosis
3. Unilateral intermittent nasal congestion with very fluid rhinorrhea
4. Intermittent exophthalmos

Subjective symptoms:
1. Unilateral orbitofrontal pains irradiating to the apex of the occipital region, descending sometimes on the right side along the neck to the shoulder
2. Photophobia
3. Inability to read longer than 5 minutes, in spite of good visual acuity with correcting lenses
4. Extreme ocular pain with the eye movements during the attacks

Repeated examinations during the eight years, with the participation of fifteen physicians and including several examinations by otorhinolaryngologists, always resulted in negative findings, until in 1947 it was discovered that the patient was affected with a chronic ethmoidomaxillary sinusitis on the right side. At this time, the patient was relieved from pain for a short time when the sphenopalatine ganglion was touched with Bonain's fluid, seeming to indicate that the patient was affected with a syndrome of Charlin-Sluder or affection of the vegetative system accompanying the second branch of the trigeminus. However, the relief was brief, and the attacks recurred more often and became more severe. All of the possible conservative treatments tried in this case failed to relieve the painful symptoms. Particularly interesting from our point of view is the fact that the mild congestion of the nasal mucosa observed by the otorhinolaryngologists was considered trivial as a causative factor of the syndrome, the otorhinolaryngologist refusing to intervene surgically. Finally, upon the insistence of the ophthalmologist, an ethmoidectomy and sphenoidectomy on the right side were performed, resulting within a week in the complete disappearance of all the irritating symptoms, enabling the patient to read without discomfort for several hours.

The authors arrive at the conclusion that in the presence of such a stubborn sensitive vegetative irritative syndrome, it is absolutely necessary to examine systematically all the sinuses, and in the presence of a chronic sinusitis, sometimes so mild that the rhinologist often refuses to operate, the ophthalmologist alone must pose the indication for nasal surgery.

In 1951, A. E. Maumenee[3] carried out homokeratoplasties on rabbits' eyes. When at the end of two weeks the transplant remained transparent, he inserted a piece of skin of the same donor into a pocket flap under the recipient abdominal wall. With this technic, the transplants, which without the inclusion of skin would have become cloudy in only one out of 60 grafts, developed cloudiness within two to three weeks after the

insertion of the skin in 28 out of 30 eyes. Maumenee concluded that his experiments proved that:

1. An opacification of corneal graft can be produced in rabbits by a donor recipient sensitization.

2. The clinical appearance of the reaction in these eyes is very similar to the clouding of mechanically successful corneal transplants in man, where the graft becomes opaque two to six weeks after the operation.

3. The clouding of corneal grafts produced in his experiments by the supplemental transplantation of the skin is further proof that the destruction of homotransplant material is the result of donor-recipient sensitization. It also demonstrates that the allergy so produced will affect more than one type of tissue from the same animal.

The statement of Maumenee can be criticized in that he used vascular tissue to sensitize the animals. This vascular tissue may produce, as he found, an allergy affecting more than one type of tissue from the same animal, including the cornea, but his experiments would have been more applicable to the problems of keratoplasty if, instead of using skin antigen to sensitize the recipient, he would have used corneal tissue.

In my clinical experience, I do not recall a single case of immediate or late opacification of the graft in favorable eyes with vascular corneas such as keratoconus that could be explained on any other basis than bacterial allergy.

Nowhere is the interrelationship between the orbital contents and the paranasal sinuses more evident than in the effect of corneal surgery or corneal traumatism, either chemical or mechanical, upon the paranasal sinuses. On the other hand, the effect on the ocular condition of the establishment of adequate paranasal sinus drainage is even more startling. It seems almost elementary to point out that in many cases the paranasal sinuses surround the orbital contents for almost three-fourths of the circumference. The intimate relation between the orbital contents and cavities which are almost universally infected and subject to constant re-infection should be quite obvious. It was this thought, and the failure of other conventional foci, that led me to do a more thorough evaluation of the paranasal sinuses. Of course, the surgery of the paranasal sinuses poses many problems which must be overcome, and perhaps this has been a factor in the avoidance of the entire issue. For instance, how adequate can intranasal ethmoidectomy be when the patient has large supraorbital extensions, perhaps large frontals, perhaps multilocular sphenoids. It must be admitted that the possibilities which such anatomy opens up are a little discouraging in the attainment of the desired results. However, by and large, results are obtainable if drainage is established according to general surgical principles. The lateral ethmoid wall and the mesial wall, for example, consist of only one thin bony partition, which in many cases is not complete throughout. The transmission through this thin wall, or by direct contiguity where this wall is absent, of bacterial toxins from the infected ethmoids to the orbital contents can be supposed to take place with great ease, especially through the apertures for the transmission of blood vessels and nerves. Such transmissions may make themselves evident only when some complicating ocular condition arises, while the original cause in the sinuses is apt to be overlooked and is subsiding in the interim of the transmission.

Obviously, the eradication of the sinus focus is the only means by which the infection or the bacterial antigens, if you will, can be kept from constantly re-infecting the orbital tissues.

CONCLUSIONS

1. In my own experience based on a great many clinical cases, of which those reported are merely examples, the al-

lergy involved seems to be of a bacterial nature.

2. The paranasal sinuses should be the most logical focus for ocular complications because of their proximity to the orbit and its contents.

3. Once the ocular complication has been established, the causative agent must be immediately neutralized by protein therapy, antibiotics, antihistaminics, cortisone and similar preparations. This failing, the causative agent must be thoroughly eradicated before irreversible changes have taken place.

4. The longer the interval between the onset of the ocular complication and the removal of the causative agent, the less can be expected from even adequate treatment.

5. The neutralization or eradication of the causative agent, in my experience, has given most gratifying results in the relatively simple complications, such as keratitis, iritis and cyclitis. Less gratifying are the results when the involvement of the inflammatory process includes several of the ocular structures.

6. There seems to be no doubt that bacterial allergy plays a very important rôle in the development of ocular complications. The mechanism whereby this takes place, I would prefer to leave to the allergist. For our purposes, what seems to be more to the point is to find the causative factor in the production of this bacterial allergy. In my experience, by far the preponderance of possible foci resided in the paranasal sinuses.

7. In the course of my experience in keratoplasty, I have found the cornea to be an extremely sensitive barometer of the presence of infection, past or present, in the paranasal sinuses. In the absence of other causes, it is the duty of the ophthalmologist to enlist and insist on the aid of the otorhinolaryngologist. On the strength of the ocular signs alone, the indication for operative otolaryngologic intervention should be established.

I fully realize that much that I have said is still of a controversial nature. If I have succeeded in raising a few doubts in the mind of the otolaryngologist as to what constitutes negative or positive findings in nasal pathology in the presence of ocular manifestations, I will feel that this presentation has been fully justified.

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EXTERNAL EYE AND AQUEOUS

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I SHALL present in this paper a brief review of the clinical work that has been collected from a number of ophthalmologists over the past twelve years in the following subjects:

1. Cytology of chronic simple allergic conjunctivitis
2. Chronic recurrent marginal blepharitis
3. Vernal conjunctivitis
4. Phlyctenular keratoconjunctivitis
5. Diagnostic anterior chamber puncture
6. Diagnostic vitreous chamber puncture

Due to the brief time allotted, only the high points will be touched upon; and, in endeavoring to keep the paper brief, many details of less important nature will be omitted.

CYTOLOGY OF ALLERGIC CONJUNCTIVITIS

A large series of patients were studied while being treated for chronic conjunctivitis in various eye clinics of St. Louis and St. Louis County and in several private offices in this area.

In each case a complete allergic history was taken. Skin and conjunctival tests for possible allergens were made along with pertinent bacteriologic procedures. Slit lamp examinations of the cornea and conjunctiva were made. Cases were selected where no specific causative agent was found, and the conjunctival smears showed a high eosinophilic count.

Method of Preparing Slides

The best smear we have been able to obtain for ophthalmologic purposes is the scrape smear. A feather-edge scraper with a straight flat aluminum edge was found to be the best. The scraper is flame sterilized just prior to use. This cleans the blade well enough for cells to cling to it quite firmly. Scrapings from the upper and lower lids are taken. After the slides are smeared, they must be properly stained. We use Hansel's stain technic.

Examination of Slides

Examination of the slides is done under the low power of the microscope, and all cells are counted, with the proportion of eosinophils to total cells recorded as a percentage figure. The higher powers of the microscope are utilized to determine the presence of bacteria, peculiarities in individual cells, and other information that may be of value.

With a properly made scrape smear, no erythrocytes should be seen on the slide. While counting cells at low power and examining cells at high power, watch should be kept for material that might represent the allergen or irritant substance.

Evaluation of Treatment

The patients in this series had an average of 3 per cent to 10 per cent eosinophils on scrape smears. The following medications were used and the results of treatment tabulated (table I).

From the department of ophthalmology, Washington University School of Medicine and the Oscar Johnson Institute.

Presented at the meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, Oct. 19, 1951, Chicago, Ill.

TABLE I

MEDICATIONS USED AND THE RESULTS OF TREATMENT IN A SERIES OF CASES OF ALLERGIC CONJUNCTIVITIS

MATERIALS	PATIENTS IN GROUP	EOSINOPHIL COUNT PERCENTAGES		SYMPTOMS	
		BEFORE	AFTER	BEFORE	AFTER
1. Zephiran, 1:5M, with Adrenalin locally every four hours	10	3-8	2-5	+++++	+++++
2. Metaphen, 1:5M, with Adrenalin locally every four hours	10	4-10	2-4	+++++	+++++
3. Antistine Ophthalmic, 0.5 per cent, locally every four hours	10	4-11	1-3	+++++	++++
4. Histadyl Ophthalmic, 0.5 per cent, locally every four hours	10	4-9	1-3	+++++	++++
5. Pyrabenzamine Ophthalmic, 0.5 per cent, locally four times a day	10	3-10	1-3	+++++	++++
6. Pyrabenzamine, 50 mg. T.I.D.	10	3-8	3-7	+++++	++++
7. Benadryl, 50 mg. T.I.D.	10	3-10	3-10	+++++	++++
8. Chlor-Trimeton, 4 mg. T.I.D.	10	4-9	4-8	+++++	++++
9. Dust Extract, 1:1,000,000, 0.2 cc. five times at 5-day intervals subcutaneously	10	5-11	2-6	+++++	++++
10. Combined Dust and Mold Extracts, 1:1,000,000 Dil. each, 0.2 cc. five times at 5-day intervals subcutaneously	10	3-9	2-5	+++++	+++
11. Staph. Toxoid, Lederle #1, 0.2 cc. five times at 5-day intervals subcutaneously	10	4-9	2-4	+++++	++
12. Staph. Toxoid, Lederle #1, 0.4 cc. five times at 5-day intervals subcutaneously	10	3-8	2-4	+++++	+++
13. Staph. Toxoid, Lederle #1, 0.6 cc. five times at 5-day intervals subcutaneously	10	4-9	2-4	+++++	+++
14. Cortisone, 6.25 mg. per 1 cc., drops every 2 hours	10	4-10	1-2	+++++	±
15. Cortisone, 3.125 mg. per 1 cc., drops every 2 hours	10	3-9	1-2	+++++	+
16. Cortisone, 1.5 mg. per 1 cc., drops every 2 hours	10	4-9	2-4	+++++	+++
17. Cortisone, 6.25 mg. per 1 cc., drops every 4 hours	10	5-8	1-3	+++++	±
18. Cortisone, 3.125 mg. per 1 cc., drops every 4 hours	10	4-10	1-3	+++++	+
19. Cortisone, 25 mg. orally four times a day	10	4-9	2-4	+++++	++
20. Cortisone, 35 mg. three times a day intramuscularly	10	5-8	1-3	+++++	+
21. ACTH, 25 mg. four times a day intramuscularly	4	5-8	2-5	+++++	++++

Conclusions

1. In chronic allergic conjunctivitis, cortisone used locally in the eye as drops or given systemically caused a greater drop in the eosinophil count on scrape smears from the conjunctiva and gave better subjective improvement than other therapeutic agents used.

2. Staphylococcus toxoid, hypodermically, was the next most effective agent in causing a drop in the eosinophil count on conjunctival scrape smears.

3. It is extremely important that in infected cases cortisone should not be used as it acts as a spreading factor,

CHRONIC RECURRENT MARGINAL BLEPHARITIS

1. ULCERATIVE
2. NONULCERATIVE

Primary Factor
(1) Hypersensitivity to Staphylococcus

Secondary Factors
(1) Seborrheic Type Skin
(2) In Ulcerative Type—Secondary Infection with Staph. Aureus or Albus

particularly in epidemic keratoconjunctivitis and acute pneumococcic conjunctivitis. We have found that in cases where the conjunctival scrape smear did not show at least 3 per cent eosinophilia, cortisone was of little value. It should be used only when the scrape smear shows we are dealing with a definite allergic factor.

CHRONIC RECURRENT MARGINAL BLEPHARITIS

Chronic recurrent marginal blepharitis is one of the very common chronic inflammations of the margins of the lids seen in clinics and private practices. It occurs in two forms, nonulcerative and ulcerative. There is always a thick reddening of the lid margins with scaling or crust formation. In most cases where conjunctival smears and lid margin smears were taken, it was found that there was a bacterial allergy and, frequently, an eosinophilic count of greater than 3 per cent.

The various types of treatment used and the results are recorded in table II.

TABLE II

RESULTS OF TREATMENT IN 282 CASES OF CHRONIC RECURRENT MARGINAL BLEPHARITIS

CASES	TREATMENT		PERCENTAGE IMPROVED
	Local *	*Systemic*	
37	Local antiseptics and 10 per cent boric unguentum	Antihistamine drugs	21
41	Antistine and 10 per cent boric unguentum	Antihistamine drugs	29
43	Local antiseptics and 10 per cent boric unguentum	Dust and mold injections—0.1 cc. of 1:1,000,000 dilution every 5 days for five doses	40
38	Local antiseptics and 10 per cent boric unguentum	Autogenous vaccines	49
23	Aqueous Cortisone (Dil. 1-4) every 2 hours for 72 hours, then every 4 hours	None	58
43	Local antiseptics and 10 per cent boric unguentum	Staph. toxoid injections—0.1 cc. Lederle's ∄1 dilution every 5 days for five doses	61
57	Local antiseptics and 10 per cent boric unguentum	Dust and mold injections—0.1 cc. of 1:1,000,000 dilution every 5 days for five doses. Staph. toxoid injections 0.1 cc. Lederle's ∄1 dilution every 5 days for five doses.	64

*All patients were instructed to clean the lids thoroughly each night and morning.

Conclusions

Best results in the treatment of chronic marginal blepharitis were obtained by:

1. The use of local antiseptics such as aqueous zephiran, 1:5000, with a little adrenalin, or aqueous metaphen, 1:5000, with a little adrenalin, used three times a day, and boric acid unguentum locally with staphylococcus toxoid, and dust and mold injections subcutaneously.

2. Similar treatment without the dust and mold injections was almost as effective.

3. Cortisone, 1:4 dilution, used locally every two hours gave very satisfactory results.

VERNAL CONJUNCTIVITIS

Vernal conjunctivitis is a chronic recurrent bilateral interstitial inflammation of the conjunctiva of seasonal incidence and unknown etiology. It is characterized by flat-topped papules, usually on the tarsal conjunctiva, resembling cobblestones in appearance; a gelatinous hypertrophy of the limbal conjunctiva, either discrete or confluent, accompanied by corneal involvement. It is associated with itching, redness of the eyes, lacrimation and a mucinous discharge usually containing eosinophils.

The cases have been divided into groups according to the type of medication used, and the results of treatment are presented in table III.

Conclusions

1. In vernal conjunctivitis, aqueous cortisone, 1:4 dilution used locally in the eyes, gave the best results.

2. Local antiseptics with endocrine therapy and staphylococcus toxoid injections also gave very good results.

3. Local antiseptics with endocrine therapy resulted in improvement in more than half the cases.

PHLYCTENULAR KERATOCONJUNCTIVITIS

Phlyctenular keratoconjunctivitis has a characteristic nodular appearance and is an affection occurring mainly in children. It is an allergic response by the corneal and conjunctival epithelium to some endogenous toxin to which the patient has become sensitized.

VERNAL CONJUNCTIVITIS

Primary Factors	*Secondary Factor*
(1) Hypersensitivity to Various Allergens especially (a) Pollen (b) Contact Allergens	(1) Endocrine Factor

TABLE III

RESULTS OF TREATMENT IN 198 CASES OF VERNAL CONJUNCTIVITIS

CASES	TREATMENT		PERCENTAGE IMPROVED
	Local	*Systemic*	
39	Local antiseptics with adrenalin	Antihistamine drugs	31
33	Antistine	Antihistamine drugs	33
31	Local antiseptics with adrenalin	Dust and mold injections	46
39	Local antiseptics with adrenalin	Endocrine therapy*	57
32	Local antiseptics with adrenalin	Endocrine therapy* and Staph. toxoid injections	74
24	Aqueous Cortisone (Dil. 1-4) every 2 hours for 72 hours, then every 4 hours	None	82

*Estrogenic hormones in females and Testosterone in males.

PHLYCTENULAR KERATOCONJUNCTIVITIS

Primary Factor
(1) Hypersensitivity to bacteria
especially (a) Myobacterium tuberculosis
(b) Streptococcus

Secondary Factors
(1) Unhygienic environment
(2) Dietary deficiency

This group of cases has been divided according to medication used, and the results of treatment are recorded (table IV).

Conclusions

1. In phlyctenular keratoconjunctivitis, aqueous cortisone, 1:4 dilution used locally in the eyes, gave the best results.

2. Local antiseptics with dust and mold injections or staphylococcus toxoid injections gave almost as good results as cortisone.

GENERAL CONCLUSIONS OF FOREGOING SECTIONS

1. *All these conditions have a basic allergic factor with added secondary etiologic factors peculiar to each.*

2. *The allergic factor in each of these diseases is influenced favorably by the use of cortisone locally or by the block action of staphylococcus toxoid or dust and mold injections.*

Cases reported in the foregoing sections were collected from a number of ophthalmologists over a period of twelve years from all parts of the United States, North Africa (particularly Egypt), Europe (particularly Italy and Southern Countries), Mexico, and South America (particularly Brazil).

DIAGNOSTIC ANTERIOR CHAMBER PUNCTURE

Among important ophthalmological problems, not one has been studied more fully and patiently than that of the aqueous humor. In our study of nongranulomatous uveitis, we have performed many anterior chamber punctures. Our technic followed that of Professor M. Amsler of Zurich, Switzerland, very closely. We used a Bowman-like needle similar to the regular twenty-six gauge needle. It has an extremely sharp arrowhead point and is fitted to a tuberculin syringe. After the well-anesthetized eye is firmly fixed by an assistant, the puncture is performed near the limbus and .1 to .2 milliliter is withdrawn. The operation, in our experience, is without danger. Of course every precaution must be taken against possible outside infection.

The following routine procedure is then carried out:

a. One drop is used for the precipitation of total protein.

TABLE IV

RESULTS OF TREATMENT IN 186 CASES OF PHLYCTENULAR KERATOCONJUNCTIVITIS

CASES	TREATMENT		PERCENTAGE IMPROVED
	*Local**	*Systemic*	
26	Local antiseptics with adrenalin	Tuberculin injections	21
43	Local antiseptics with adrenalin	Autogenous vaccine injections	31
34	Local antiseptics with adrenalin	Staph. toxoid injections	37
24	Local antiseptics with adrenalin	Staph. toxoid injections Antihistamine drugs	42
32	Local antiseptics with adrenalin	Dust and mold injections	47
27	Aqueous cortisone (Dil. 1-4) every 2 hours for 72 hours, then every 4 hours	None	51

*Atropine used locally in most cases.
Cases in this table were collected from a number of ophthalmologists.

TABLE V

RESULTS OF TREATMENT OF CASES OF LENS ANAPHYLAXIS AND NONGRANULOMATOUS UVEITIS

DIAGNOSIS	CASES	PERCENTAGE OF EOSINOPHILS			TREATMENT*	RESULT
		BEFORE TREATMENT	4TH DAY AFTER TREATMENT	10TH DAY AFTER TREATMENT		
Lens Anaphylaxis	1	24	16	2	Removal of Lens Cortex	Improved
Lens Anaphylaxis	1	18	8	2	Removal of Lens Cortex	Improved
Nongranulomatous Uveitis: Streptococcus Hypersensitive	5	4-8	3-7	2-5	Streptococcus desensitization Fever (Typhoid)	3 Improved
Nongranulomatous Uveitis: Nonspecific	5	4-12	4-8	2-5	Cortisone, Dil. 1-4, every 2 hours	3 Improved
	3	5-13	5-11	1-3	Fever (Typhoid)	2 Improved
	4	5-15	4-8	2-3	Fever and Cortisone	All Improved
	11	0-2	0-1		Cortisone, Dil. 1-4, every 2 hours	3 Improved
	14	0-2	0-1		Fever (Typhoid)	11 Improved

*Atropine used locally in all cases.
Cases reported in the above chart were collected from a number of ophthalmologists.

b. One drop goes in the Fuchs-Rosenthal counting cell chamber.
c. One drop for bacteriologic culture.
d. Two or three drops are centrifuged in the capillary tube of the Verry apparatus and provide us with identification of the cellular elements.

The results of treatment, following anterior chamber puncture, are recorded in table V.

Conclusion

1. In nongranulomatus uveitis, local cortisone is of definite value if the anterior chamber eosinophilia is 4 per cent or more. If the eosinophilia is less than 4 per cent, cortisone has little value.

DIAGNOSTIC VITREOUS PUNCTURE

Another important ophthalmologic problem is the diagnostic vitreous puncture. At this time I would like to report a group of 19 cases of blind eyes which were in the advanced stages of degeneration. A diagnostic vitreous puncture was made and smears examined for eosinophils before enucleation. A twenty-four gauge needle, well beveled, was inserted just posterior to the ora serrata and the vitreous sucked up in the canaliculus of the needle by vacuum. This procedure was carried out after routine preoperative preparation of the eye. The vitreous obtained was then smeared for cell study using Hansel's stain, and

TABLE VI

EOSINOPHIL COUNT IN 19 CASES OF DEGENERATION DETERMINED BEFORE ENUCLEATION

CASES	CAUSE OF DEGENERATION	PERCENTAGE EOSINOPHILS
2	Old Retinal Detachments	4-8
1	Intraocular Malignant Melanoma	6
1	Epithelization of Anterior Chamber after Cataract Extraction	16
4	Trauma	5-12
11	Old Uveitis	2-18

Cases reported in the above chart were collected from a number of ophthalmologists.

Wright's stain. Cultures for bacteriologic study were also made and total protein determinations carried out (table VI).

Conclusion

1. It is my belief that an allergy to toxic protein material liberated by the degenerating tissue is a determining factor in the rapidity and extent of the development of phthisis bulbi in these cases.

I wish to express my appreciation to the number of ophthalmologists, too numerous to mention individually, who were so kind in sending me their case data. I particularly want to thank Dr. French K. Hansel for his valuable aid.

DISCUSSION

CONRAD BERENS, M.D., New York, N. Y., Moderator: I have a question for Dr. Hanser. "What substance is used in streptococcus desensitization?"

DR. HANSER: Streptococcus vaccine was made up according to the patient's individual streptococcus sensitivity. Dosage was calibrated according to the individual concerned and his reactions. However, the injections were given at three- to five-day intervals as Dr. Alan Woods reported in his paper about a year and a half ago. I am sorry I cannot give you a definite answer.

DR. BERENS: Thank you. There is another for you from Dr. Mazique. "Your report shows a high percentage of improvement with cortisone. What about the duration of improvement and the advisability of continued use?" Do you care to answer that, sir?

DR. HANSER: That is a very interesting question because patients have to be kept on cortisone for varying lengths of time. They will have intervals when they will be free of their symptoms and you tell them to go along without their drops but to use them as they find need for them. Some of these people that we have reported, particularly vernals, will have seasonal incidences when they will use their drops four, five or six times a day. Then they will go along for a number of weeks without using their drops at all, but many of the cases are still on the drug. It cannot be completely removed in all cases.

DR. PREWITT: I have three or four questions I want to ask. In his book Dr. Sluder mentioned edema due to emotional tension, and then two years ago here Dr. Wolff mentioned the marked edema that a patient gets due to emotional stress and strain, which is classified as edema psychogenesis. On that basis, might I ask Dr. Castroviejo, "Did any of these cases subjecting themselves to a corneal transplant or to any operative surgery, preoperatively get an emotional edema and light up an old bacterial infection that constitutionally was allergic, the eye being the flare organ for the reaction?"

DR. CASTROVIEJO: I came to this meeting hoping that I could ask that question myself, because there are many things in my mind that are not quite clear. The explanation that you just gave seems to be reasonable. I cannot give any explanation. I have just stated facts, what I have been able to observe without being able to interpret. I have tried in the paper to interpret some of the clinical facts that have been presented. I did not have time to read the entire paper and you may be able to find some answers in it when it is printed. But for many questions I have no answers.

DR. HIGGINS: What is the distinction between focal infection and allergy?

DR. BERENS: The subject of focal (chronic) infection has interested us for over thirty years, and we have spent much time trying to find out what we could about it. As far as our experimental work is concerned, we thought we had what were known as toxicity tests, and they are rapidly and generally being used for staphylococci. The tests we use to determine the "toxicity" of alpha hemolytic streptococci are not generally used. These findings are valid with 92 per cent correlation with animal inoculation.

However, the worst part of this is that when we inject these so-called toxic organisms into animals the first time, we get no more uveitis with the "non-toxic" than we do with the "toxic" organisms. That work was published some years ago. The "toxic" organisms seem to make better bacterial antigen. So much for that part of toxicity.

However, although we know much from animal experimentation—I do not go so far as to translate animal experiments to man—I do not think we have proved focal infection in man. That is the reason we are still working on the problem. So far as animal inoculations are concerned, if you repeat them with the same organism which produced no inflammation (which often occurs), some of these animals become sensitized. (The group that I worked with did not use the word "sensitized." They used the word "immunized.") Then inflammation will develop in the sensitized animals. That is one of the reasons

I believe that allergy plays a significant role in this question of chronic (focal) infection.

DR. BERENS: Dr. Hanser has been asked another question. "Have you had any experience with filling the anterior chamber with cortisone in anterior uveitis?" This question is asked by Dr. S. B. Forbes of Tampa, Fla.

DR. HANSER: No, we have not injected cortisone into the anterior chamber. However, in many cases of anterior uveitis we do inject adrenalin directly into the anterior chamber to break up the adhesions of the iris. This works extremely well.

DR. BERENS: Thank you very much, Dr. Hanser. Has anyone in the audience had experience with this method of treatment? I have had no experience with it. Some of the men this morning spoke about using it in the nose for a year without producing difficulties.

I have another question for Dr. Hanser. "Have you noted any allergy or allergic manifestations to cortisone per se?" Dr. Gordon Vancil asks the question.

DR. HANSER: We have not. However, Dr. Stanley Hampton, of St. Louis, has reported two such cases.

Here is another question, from Dr. Jack Cowen of Chicago, Ill. "What further relationship have you found concerning allergy in chalazia?"

I do not know how to answer this question, but in many cases where the cultures show a Staphylococcus albus or Staphylococcus aureus infection, Staphylococcus toxoid or autogenous vaccines do help. The case history of the patient certainly should be thoroughly worked up from every standpoint, including the refractive error. I cannot answer any further than that.

DR. THOMAS D. ALLEN: I think Dr. Hanser mentioned testosterone. Will he explain what relationship testosterone has to allergy?

DR. HANSER: The situation in which testosterone was used was a case of vernal conjunctivitis in which the child showed a definite endocrine deficiency. This form of therapy started many years ago before we had sulfonamides and they were using testosterone and theelin in vaginitis. They found that in several cases of vernal conjunctivitis, endocrine therapy helped. It was in that relationship that a series of cases was followed for a number of years. The results of that study were presented today.

DR. BERENS: "Did any cases of corneal edema develop during prolonged use of cortisone?" This is asked by Dr. V. A. Chapman of Palm Springs.

DR. CASTROVIEJO: I have seen some cases of corneal edema disappear during the use of cortisone, and I have seen other cases that apparently were beginning to improve with the use of cortisone develop edema, which I have not been able to explain unless the use of cortisone may have some influence in the regeneration of the corneal nerves.

Another question, "Why does the transplanted cornea alone become cloudy?" I have been trying to get an answer to this question for myself for the past twenty-three years. I can only guess at the answer. I believe there is more than one explanation. The transplant remains for six months somewhat isolated, without really completely integrating with the rest of the organism until all the structures of the cornea have regenerated. The epithelium and the endothelium regenerate very quickly, but not Descemet's membrane. It takes six months to regenerate fully, and as long as Descemet's membrane is not present any inflammation which involves intraocular structures is likely to affect the transplant also.

Another observation that has been made on regeneration of the corneal transplant is that if you wait too long to remove the allergic antigen, corneal dystrophy takes place, with permanent corneal edema, particularly edema of the epithelium and the endothelium. I think this edema is related to degeneration of the corneal nerves. This point is discussed in my paper. I have never been able to verify the statement made by Maumenee that when a transplant becomes cloudy it is caused by a donor recipient sensitization. In my experience this has always happened in the presence of focal infection.

DR. BERENS: Thank you, Dr. Castroviejo. We have one other question. "As a practicing allergist I am embarrassed by not being able to find so easily any positive evidence of allergy in many of the cases sent to me. How does everyone find these food allergies? What is your criterion for calling those conditions food allergies?"

Several of you spoke about allergy. Do you want to answer this question?

It is a fair question. We have been very embarrassed by not being able to determine food allergies. Of course, some of you think the things one is most allergic to they eat every day. It is very hard to take the person

off that food for four or five days to make a diagnosis. We work with Bob Cook, who I think was one of the earliest men among the allergists to give consideration to the question of bacterial allergy. I think he has now proved it thoroughly to my satisfaction as well as to others. I do think it is important. I have also worked very closely with Dr. Coca, who has written a book on the pulse-diet method in studying allergy. He has given us evidence that patients who are allergic to food have an increase in their pulse rate after ingestion of the particular food. That is one way. Another way is to measure the degree of leukopenia after taking these foods that are suspected of disagreeing.

I think we would all be very greatly indebted to Dr. Berke if he would discuss some of the things that have not yet been mentioned here. Is Dr. Berke here? Will you give us a little dissertation?

I think we as ophthalmologists have had to put our necks out not only on the question of food allergy but on the question of bacteriology in the discussion of this subject.

DR. BERKE: I am associated with an eye, ear, nose and throat clinic, and many of the cases presented here today have been referred to me. I go through the routine testing of the individual by means of skin tests, take a careful history, and after a little provocation on the part of the ophthalmologist, I go ahead and check foods by elimination diets, and end up with a negative answer.

I have often wondered if the fault was mine, whether someone had something up his sleeve that I did not have, or that I had omitted something. So I presented this question, hoping that it would stimulate more discussion and throw light on the subject.

As for the use of the test created by Dr. Coca, by which increase in pulse rate after food ingestion is taken as a criterion of food allergy, I have always been a little hesitant about using it. The use of the leukopenic index in the study of food allergy is not generally accepted.

The word "allergy" has been used here today with regard to "infectious allergies," something which we do not know too much about and cannot prove. I have had the experience of making and testing vaccines of pure cultures of staphylococci, streptococci, pneumococci, influenza bacilli, and a number of other bacteria in an attempt to find out whether people reacted to them specifically. We never were able to get constant, positive, specific reactions. Incidentally, these were used in the study of "infectious" asthma; our results with treatment with these vaccines were uniformly unsatisfactory.

I have seen some good results in the treatment of eye conditions, from the use of vaccines of all types in both high and low dilutions, but I have always considered the effect "non-specific." Some, too, have cleared up after food elimination.

DR. BERENS: Thank you very much. We appreciate your contribution to our discussion here.

"A plea for consideration. The rhinologists joke about seeing cases they think normal and need to be definitely educated as to what eye men want on such an examination."

One of our group here does nose and throat work as well as eye work. We are going to ask Dr. Sayad if he will answer this for us.

DR. SAYAD: This will probably start the fight. I thought probably, having such a large crowd as this, allergy was pretty well substantiated as a definite etiologic factor. There is still a great deal of feeling among medical men as to the virtues of allergic investigations, but I think some men are still uneducated in allergy and they do not believe in it. They still think that allergic investigation is more or less an unreliable method of diagnosis. I think if eye men and ear men had more meetings of this sort and were willing to accept the views of each other and if they would compare their histories and their results, they would come to the conclusion that there is a great deal to be gained in knowing allergic manifestations. The man who does both eye and ear work and is interested in allergy does not have this controversy. I do not recommend that everybody do this, but I think that men who are doing allergy in the eye and have accepted the theory of allergy in the ear, nose and throat can get together and solve these problems.

The eye men have been very slow on the trigger with regard to allergy, and otolaryngologists are to be congratulated on having taken the lead in it. But I think that ophthalmologists have now come to the point where they accept the fact that a great many ophthalmologic conditions can be explained by allergy. I think the plea that the doctor from Oakland has made is quite timely and calls for cooperation between otolaryngologists and ophthalmologists in their approach to allergy as a method for solving a great many of their problems.

DR. BERENS: Thank you, Dr. Sayad. We have one other question but I doubt that we

have time to answer it adequately. "A recent cooperative study reported in the Journal of the American Dental Association casts considerable doubt on the whole hypothesis of focal infection and implies, in my opinion, that most remissions obtained in rheumatoid arthritis or eye inflammations, after teeth are extracted, are purely coincidental postoperative effects. Will panel please comment?"

We have worked with some of the top men in the country in trying to get somewhere on this question of dental focal infection. I really feel that we would have to spend the entire day on this to make a proper reply because I have read this report with great interest and care. All I can say is that it is a negative report. I have looked into the records of the men making the report and know they could not be a finer group. However, most of them are not practicing in the field of medicine, and while they cast complete doubt on the improvement in some of these patients treated for chronic infection, I believe they would probably change their minds if they practiced ophthalmology. This is only my personal opinion, and I hope we will have more scientific facts to bring to you in the years to come.

THE USE OF STAPHYLOCOCCUS TOXOID AND THE EXTRACTS OF PATHOGENIC MOLDS IN OTOLARYNGOLOGY AND OPHTHALMOLOGY

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STAPHYLOCOCCUS TOXOID

STAPHYLOCOCCUS toxoid has been used for the prophylactic treatment of recurrent boils, carbuncles, styes, and pustular acne since 1933. It has been suggested that the reactions noted in some cases indicate a hypersensitivity to staphylococcus proteins. The best results were noted in patients with a high sensitivity.

Standardization

Staphylococcus toxoid is prepared from a highly hemolytic strain of Staphylococcus aureus and is tested for its ability to stimulate antitoxin production. A unit of toxoid is the amount obtained from a dermonecrotizing unit of toxin (the least amount of toxin which by intradermal injection in a susceptible rabbit will produce an erythema with a central necrosis at least 5 by 5 mm. in diameter). Detoxification of the toxoid is effected with formaldehyde. It is carried to the point where the intradermal injection of 0.1 cc. into the skin of a susceptible rabbit will no longer cause necrosis.

Tolerance Test

The manufacturers (Lederle, Sharp and Dohme) recommend a preliminary toxoid sensitivity test before the institution of treatment because in rare instances local and general reactions to 10 units (0.1 cc. no. 1 dilution, 100 units per cubic centimenter) have been noted.

The sensitivity test is made by the intracutaneous injection of one unit (0.1 cc. of a 1:10 dilution of Dilution 1). At the end of 24 hours the test is read and recorded as 1+, 2+, 3+, or 4+ according to the following results:

1+ : Erythema less than 10 mm.
2+ : Erythema 10 to 15 mm.
3+ : Erythema over 15 mm. without necrosis.
4+ : Over 15 mm. with necrosis (rare).

Dosage: The initial dosage based on the above tests is as follows:

1+ reaction—0.10 cc. Dil. 1 (100u per cc.) or 0.01 cc. of Dil. 2 (1,000u per cc).
2+ or 3+ reaction—0.10 cc. of a 1:10 dilution of Dil. 1 (1 unit).
4+ reaction—0.10 cc. of a 1:100 dilution of Dil. 1 (0.1 unit).

By this plan of treatment, injections are recommended by the subcutaneous or the intramuscular route and are given twice weekly. In general, the dosage is increased by 0.2 cc. with each injection until the top dose of 1.0 cc. of Dilution 2 (1,000 units) is reached. This usually requires about 10 injections. After the maximum dosage is established, the injections are given at weekly, sometimes at three to four week intervals.

Observations on the Use of Small Doses

In the above discussion, it was stated that the best results were obtained in patients with the highest degree of sensitivity. In these instances, very small doses were used. The initial dose was as small as 0.1 unit. In the treatment of furunculosis of the ear canal and styes,

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we noted very dramatic effects with doses of from 0.1 to 0.5 unit. These results suggest that the small optimum dosage is more satisfactory and that the build-up to the maximum tolerance of 1,000 units is unnecessary.

The Use of Staphylococcus Toxoid in Allergy

It is a well-known fact in allergy that there are a number of nonspecific agents or factors which have a very striking effect on the allergic state. For example, shock or fever from operations, burns, fractures, or severe acute infections (erysipelas, pneumonia) will produce a very dramatic or complete relief of allergic manifestations of all types. The trauma of major sinus operations may effect relief rather than the drainage or removal of pathologic tissue. The relief produced by these various shocks or stress factors, according to our present knowledge of the mechanism of the allergic reaction, results from a response of the adrenergic system (stimulation of the production of ACTH and/or cortisone).

Urticaria and angioedema. About five years ago an incident occurred in the treatment of a patient suffering from an acute attack of generalized urticaria and angioedema. Because of the absence of any definite etiologic factor, it was thought advisable to try the use of some nonspecific agent for its shocklike effect. Staphylococcus toxoid was chosen and a dosage of one unit was injected intracutaneously. Within a few hours the rash and swellings showed marked improvement, and within 24 hours they had disappeared. After a period of three days there was no recurrence of symptoms, but another injection was given. No further symptoms reappeared. As subsequent cases of this type appeared, the same type of therapy was employed with a very high percentage of good results. This treatment was found to be effective also in chronic urticaria.

In all of the cases so treated, skin tests and an investigaiton of the etiologic factors were instituted after the symptoms had subsided.

Serum sickness. Later it was noted that these small doses of staphylococcus toxoid were effective in the treatment of serum sickness reactions, especially those manifested by urticaria and angioedema, and especially those resulting from the injection of penicillin. Not infrequently, only one or two injections are necessary to produce complete relief.

Allergic dermatitis, respiratory allergy and rheumatoid arthritis. Satisfactory response was also noted in atopic dermatitis, infantile eczema, contact dermatitis and seborrheic dermatitis, especially that involving the ears and the face. In some cases of nasal allergy and bronchial asthma, good results were also obtained. In small doses of 0.1 to 0.2 unit it was effective in the relief of rheumatoid arthritis.

The exact mechanism by which staphylococcus toxoid is effective is unknown. It has already been suggested that the adrenergic system is involved. In any event, it "blocks the cycle" of allergic reactions.

Dosage Recommended

The effective dosage varies with individuals and with age, as well as with the various manifestations of allergy. The range of dosage is from about 0.1 unit as the lower limit to about 1 unit as the top dose. In some instances it may vary somewhat more to the weak or to the stronger side. If the dose is too small, no effect is noted. If it is slightly on the strong side, a temporary flare of the symptoms and lesions may occur, followed by improvement. In such instances, the dosage should be reduced. If a large dose, such as 5 to 10 units, is given, there is no effect whatsoever.

TABLE I

STAPHYLOCOCCUS TOXOID

Treatment of
Urticaria, Acute and Chronic.
Post Penicillin Reactions—Serum Sickness.
Infantile Eczema. Atopic Dermatitis.
Contact Dermatitis.
Rheumatoid Arthritis—0.1 unit or less.
Furunculosis of Ear Canal, Styes, etc.
Marginal Blepharitis.

Stock Solution #1, 100 Units per cubic centimeter.

1 cc. 100 unit Sol. + 9 cc. Diluent = 10 cc., 10 units per cubic centimeter.
1 cc. 10 unit per cubic centimeter of Sol. + 9 cc. Diluent = 10 cc. 1 unit per cubic centimeter.
1 cc. 1 unit per cubic centimeter of Sol. + 9 cc. Diluent = 10 cc. 0.1 unit per cubic centimeter.

Begin treatment with intradermal injections every three or four days.
In most cases it is well to try 0.5 unit as the first injection, then increase or decrease as indicated.
Increase or decrease dosage according to the response of a few treatments, then maintain dosage at most effective point. Do not build up dosage.
If a slight flare-up occurs after an injection, reduce the dosage. After relief of symptoms, increase intervals to week or ten days. If still clear, discontinue treatment.
When symptoms recur, look for cause of exacerbation—food, contact, etc.
Results are excellent if dosage is properly calculated.
Since treatment should be given intradermally, the amounts should be small, so the following plan is suggested. Notice that the largest is .10 and the smallest is 0.02 cc.

Ten Units per cubic centimeter Urticaria, Angioedema, Penicillin, Staph. Infections, Contact Dermatitis			*One Unit per cubic centimeter* Infantile Eczema, Rheumatism, Arthritis		
1.0	Units	.10 cc.	.10	Unit	.10 cc.
.80	"	.08 cc.	.05	"	.05 cc.
.50	"	.05 cc.	.03	"	.03 cc.
.30	"	.03 cc.	.02	"	.02 cc.
.20	"	.02 cc.			

Since the initial dose which has been usually employed is 10 to 20 units, the effect of the small doses was not observed, except in those instances of a high degree of sensitivity, in which 0.1 to 1 unit was given. The good results from these doses was soon lost when they were increased out of the range of effectiveness. These observations have been made by a number of allergists, but no one apparently maintained the dosage at the optimum level.

In infantile eczema, seborrheic eczema or seborrheic dermatitis in infants and small children, the initial dose should be about 0.1 to 0.2 unit, injected intracutaneously every three to four days. As improvement progresses, the intervals may be increased to 7, 10 or 14 days. In most cases of all types in adults, the initial dosage should be 0.5 unit. Subsequent dosages should be maintained at this point if satisfactory response is noted, otherwise dosage may be changed to a higher or lower level as indicated. As a rule, if no response is noted to 0.5 unit, the dose may be increased to 0.8 or 1 unit. If there is some flare of lesions or increase in symptoms, the dosage should be decreased. The effect is much more striking than that ever observed with the large doses.

The technic of preparations of the dilutions and the amounts recommended for intracutaneous injections are outlined in table I.

In employing the intracutaneous method of administration, the amounts injected must be small. So far, no untoward local reactions have been noted.

Duration of Treatment

When the urticaria, eczema, or contact dermatitis has completely subsided, treatment may be discontinued. There may be no further recurrence. In some instances of eczema the results were good in spite of the fact that no foods were eliminated from the diet. In most

cases, however, the etiologic factors must be eliminated to prevent recurrence. If satisfactory results are not obtained early from toxoid therapy, it may be advisable to employ an elimination or trial diet. Skin or patch tests should be performed as indicated. Staphylococcus toxoid may also be used with the extracts of the pathogenic fungi, trichophytin, monilin and epidermophytin. This combination has produced good results in eczema.

TREATMENT WITH EXTRACTS OF THE ATMOSPHERIC AND THE PATHOGENIC FUNGI

The extracts of the atmospheric molds are used chiefly in the treatment of respiratory allergy, while the pathogenic molds are more frequently employed for the treatment of dermatologic conditions.

Atmospheric Molds

The atmospheric molds such as alternaria, hormodendrum, helminthosporium, rusts and smuts, acting very much like pollen, cause nasal and bronchial symptoms. The pathogenic molds, especially the monilia group, may be responsible for secondary infection in bronchial asthma. Their recognition is important. In some cases of hay fever and also house dust allergy, there is a sensitivity to the atmospheric molds. Hay-fever-like symptoms, occurring during the warm months of the year, may be caused by these molds rather than pollen. Since these molds are also included in house dust, we have routinely employed a mixture of 90 per cent house dust and 10 per cent atmospheric molds in treatment. By means of serial dilution testing, we found that the mold is about ten times stronger than the dust extract. In some instances, it may be desirable to use only the mold extract in treatment, especially in cases of vernal conjunctivitis. The mold extracts may be prepared from cultures or from the natural feed mill dust such as that suggested by Wittich. We have had several cases in which corn smut was the cause of periorbital dermatitis.

In general, the dosage of atmospheric mold extract is about the same as that of house dust.

Pathogenic Fungi

Although pathogenic fungi, especially monilia, may involve the respiratory tract, they are most commonly encountered in dermatologic conditions. The types of fungi which come under consideration are (1) trichophyton, (2) monilia and (3) epidermophyton. These fungi commonly cause lesions of the hands and feet. Lesions around the eyes and ears are also encountered. One area of involvement, such as on the feet, for example, may act as the primary focus and other distant lesions may appear as "ids." In some instances, it may be a question of whether the process is an infection, an allergy, or a combination of the two. In any event, the patient must be considered as susceptible to the infection or allergic to the fungi or their byproducts.

Treatment

The injection of fungus extracts, however, should be effective in the treatment of both conditions. We employ a mixture of trichophytin, monilin and epidermophytin (Hollister-Stier). The stock solution 1:1,000 (no. 3) is diluted serially through numbers 4, 5, 6, 7, 8, 9, and 10 (number indicates number of zeros. For example, no. 8=100,000,000). We have found that the dilution most commonly effective is number 7, or 1:10,000,000. Sometimes number 6 (1:1,000,000) or number 8 (1:100,000,000) is more satisfactory. In extreme cases, number 9 or number 10 may be used. For a nonspecific blocking effect on the allergic process or for staphylococcus immunization (secondary infection), staphylococcus toxoid may be added to the injection of fungus extract.

TABLE II

TRICHOPHYTIN-MONILIN-EPIDERMOPHYTIN

Stock 5 cc. 1:1,000 Solution Glycerinated

Hollister-Stier Laboratories
727 Penn Avenue
Wilkinsburg, Pa.

	NUMBER
.10 cc. 1:1,000 + .90 cc. Saline = 1 cc. 1:10,000	4
.20 cc. 1:10,000 + 1.80 cc. Saline = 2 cc. 1:100,000	5
1.00 cc. 1:100,000 + 9.00 cc. Saline = 10 cc. 1:1,000,000	6
1.00 cc. 1:1,000,000 + 9.00 cc. Saline = 10 cc. 1:10,000,000	7
1.00 cc. 1:10,000,000 + 9.00 cc. Saline = 10 cc. 1:100,000,000	8

Begin treatment with .10 cc. 1:10,000,000 (No. 7) every five to seven days.
Increase or decrease dosage according to response of few treatments, then maintain dosage at most effective point. Do not build up dosage.

Indications: Fungous allergy or infection of ear canal, auricle and surrounding areas, periorbital dermatitis, athlete's foot, etc. May also use staphylococcus toxoid for combination of allergy and fungous infection.

Results of Treatment

The results obtained in the use of small optimum doses has been most dramatic. The itching usually ceases almost immediately and the lesions begin to heal promptly. These responses indicate that immunologic protection can be obtained by these small doses. It is important to emphasize that the effective dosage must not be changed. There must be

Reports in the literature on the use of injectable fungus extracts, on the whole, indicate that the results have not been satisfactory. Most observers, especially the dermatologists, have discarded them. It is evident that failure to obtain satisfactory results is caused by overdosage. An initial dose of 1:1,000, 1:10,000, or even 1:100,000 is much too strong. Initial small doses of this type, however, may be effective in some cases, but a satisfactory response may be lost by increasing the dosage.

A great many cases of dermatitis are encountered in which it is a question of atopic dermatitis, contact dermatitis or a fungus (allergy or infection). Prompt initial improvement may be obtained by the use of staphylococcus toxoid, fungus extract or a combination of both. In the meantime, the elimination or avoidance of the etiologic factor may be investigated.

If a combination mixture of staphylococcus toxoid and the pathogenic molds is desired, it may be prepared as follows:

1. Dose of Staph. Tox. for .10 cc. = 0.50 unit.
2. Dose of T.M.E. Molds for .10 cc. = .10 cc. no. 7 (1:10,000,000)
3. Total cubic centimeters needed = 10.
4. Units of Staph. Tox. = 50 or .50 cc. no. 1 Dil., 100 units per cubic centimeter.
5. Amount of T.M.E. Molds = 1.00 no. 6 Dil. (1:1,000,000).
6. Amount of diluent sufficient for 10 cc. = 8.50 cc.

These doses represent an average initial amount.

If a weaker solution is desired, .20 unit of staphylococcus toxoid may be given with .10 cc. of number 8 T.M.E. Molds (1:1,000,000,000). For 10 cc. of solution = 20 units of staphylococcus toxoid or .20 cc. of the number 1 dilution (100 units per cubic centimeter), plus 1 cc. of the number 7 dilution of T.M.E. Molds, plus 8.80 cc. of diluent.

In the treatment of bronchial asthma in which the pathogenic molds may be a factor, they may be administered as such or combined with house dust.

EXPERIENCES WITH ALLERGIC CONJUNCTIVITIS AND DERMATITIS OF THE LIDS

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JOPLIN, MO.

THE allergic individual comprises 10 per cent of the population. Positive reactions of the skin or mucous membranes to specific allergens are obtainable in over 50 per cent of the cases.[1] It is no wonder then that ophthalmologists are frequently called upon in every-day practice to treat allergic conjunctivitis and dermatitis of the lids. These conditions are vexing to the physician and extremely discomforting to the patient.

Woods[5] has summarized the various common lid and conjunctival types as (1) that seen in hay fever and asthma and characterized by sudden onset with marked chemosis and edema, absence of bacteria and purulent secretion, and (2) the eczematous type, typically seen after the use of a drug to which the patient is sensitive. This is the type most often seen. It is accompanied by intense itching and lacrimation with redness of the skin of the lids. The latter has a parchment-like feel which is characteristic. Often the skin is fissured where the secretion exudes onto the skin.[4] Chronic conjunctivitis is probably the commonest of all types and is not often recognized as being allergic in origin. Dryness and itching are usual complaints and blepharitis is common.[5] Vernal catarrh is characterized by either palpebral or bulbar follicular hypertrophy, seasonal incidence and a peculiar slimy secretion. Its allergic assumption is based on its incidence in the young, seasonal occurrence, absence of bacteria and abundance of eosinophils in the conjunctival secretion.

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Before submitting my case reports it is pertinent to outline briefly practical office procedure in approaching this problem. The history will often furnish the clue as to the primary cause: Where was the patient and what was he doing before the onset of symptoms? Was he, for example, driving cross country, down on the river, mowing the lawn, cleaning house or moving from one house to another? What about new facial contacts such as new synthetic soaps, cosmetics, perfumes, nail polish? Were there new occupational contacts?

The number one suspect will be the inhalant of the season during which the condition first started. If a patient suddenly begins to react to a cosmetic or other contact that he has used for some time with impunity, one should always suspect and test for the inhalant or inhalants associated with the season at which time the trouble began. In other words, something had to raise the allergic state of the individual and therefore cause him to react to substances that ordinarily would not cause clinical symptoms. This same principle has been observed for years with foods which will cause clinical symptoms only during a pollen season that the patient reacts to, and the rest of the year the food can be taken with no symptoms whatever.

There are many cases which, once they get started, will not clear up by merely eliminating the contacts unless the inhalant of that season is also dealt with. Consequently the inhalants are tested for and specific treatment given when indicated, along with the usual elimination of suspected local contacts. Of course the starting of this specific

treatment might be a little slow, so a nonspecific treatment is begun the same day as the inhalant tests are given. The inhalant tests are usually only two or three: dust molds and the pollen, if any. Very weak tests are given. Begin with a 1-unit dose of staphylococcus toxoid subcutaneously and repeat the dose at two-day intervals for three doses. Usually marked improvement has occurred by this time and the nonspecific therapy has produced a blocking effect on the allergic reaction. On the sixth day a desensitizing dose of the inhalant is given, which is usually house dust, molds and the specific pollen. These must be given in low desensitizing doses because, if overdosed, a reaction will occur and a recurrence of the acute symptoms will appear. However, if the dose is correct, the relief from symptoms will continue to be even better. The staphylococcus toxoid is then given three days later and the desensitizing dose and toxoid repeated again in six days. If the symptoms have by this time completely subsided, the patient returns in six days for the injections. If there is a slight flare-up after the specific therapy, the dosage is reduced back to a relieving dose.

Case 1. Mrs. R.K. reported on February 23 for treatment. She complained of itching and burning of the lids and eyes for several weeks. Examination revealed a chronic marginal blepharitis and conjunctivitis. She was advised to omit all cosmetics and facial contacts except lipstick for two weeks. One unit of staphylococcus toxoid was given subcutaneously. She returned in one week, much improved, but some symptoms remained. She was tested for dust and molds, cigarette smoke and tree pollen, which all gave good reactions. Staphylococcus toxoid, ½ unit, was given. For several days after these tests her symptoms were worse because the test doses were too strong. However, she was almost completely relieved after treatment with her first desensitizing dose; and after several weekly injections, she was dismissed completely well.

Case 2. Mr. L.M. reported for treatment December 1, with a history of severe itching, burning, swelling, photophobia and lacrimation of each eye for three months. Treatment by three physicians had brought no results. He had been working in the hay fields and weeds at the time symptoms first appeared. Smear of eye secretion was four plus eosinophils and two plus neutrophils. He was tested for dust, molds, ragweed, weeds, and tall grasses. At the same time he was given 1 unit of staphylococcus toxoid. He returned two days later much improved. In fact, he felt great relief the first evening after the toxoid and intradermal tests. Treatment doses of the allergens plus the staphylococcus toxoid were given every six days for five visits with complete relief of symptoms.

Case 3. Mrs. D.H. reported for treatment December 18, complaining of thickening and itching of the lids for five months. Examination revealed thickened, brawny skin of the lids—a typical allergic dermatitis. Nasal smear revealed three plus eosinophils. All cosmetics and facial contacts were eliminated. Intradermal tests for dust, molds, mixed weeds, grasses and orris root were all positive. One unit of staphylococcus toxoid was given subcutaneously. The patient returned December 23 practically asymptomatic. The swelling and edema had almost subsided. Treatment doses of house dust, air-borne mold, tobacco smoke, ragweed and orris root were given. After two additional treatment doses she was completely well and has remained so.

Case 4. Mrs. V.S. had suffered for two weeks with intense itching and burning of the eyes. There was marked brawny thickening of the skin of the upper lids. Treatment by a dermatologist for two weeks had brought no relief, and her symptoms had progressively became worse, although all contacts of cosmetics, soaps and fingernail polish had been completely withdrawn. She gave a history of ragweed hay fever, but had not had an attack for seven years. An eye smear was positive for eosinophils. Intradermal tests to tree pollen and tobacco smoke were positive. Tree pollen was the most suspected offender as the season was in full swing at this time. One unit of staphylococcus toxoid was given on March 24. On March 27 she was much improved and comfortable. She was given one unit of staphylococcus toxoid with low dosage hyposensitization to trees and tobacco smoke. The treatment was continued every four days through April and, in the meantime, she was found to be sensitive to grasses, whereupon

this treatment was also incorporated. She was discharged June 12 completely well and has had no recurrence.

Case 5. Mrs. L.K. was seen on October 28, 1949. She complained of "eye allergy" which had been present constantly for four years in the form of a rash on the forehead and lids, with marked brawny thickening of the skin of that area. The same type of lesions existed on the elbow and hands. She had been treated by five different doctors for the condition with absolutely no improvement, although she had been given all the usual lines of treatment. I remembered a case of severe allergic dermatitis in a dog, described to me by Rinkel,[2] which he had been able to keep under control by house-dust desensitization or by removing the dog from contact with house dust. Using this principle, the following course of therapy was carried out: She gave a strong positive test to house dust and molds. Ragweed was negative in all dilutions. She was given one unit of staphylococcus toxoid. In one week she was much improved; then she was given staphylococcus toxoid and low dust and mold injections once a week until December 30. At this time she was completely well. Even the elbows and hands were completely clear. On October 19 of the following year she returned with a slight recurrence, complaining of itching of the nose and a rash on the right elbow. At this time it was found that eggs would cause itching of the skin on clinical feeding tests, and she was easier to control after they were omitted from the diet. Staphylococcus toxoid and dust and mold injections were again given with prompt relief of the symptoms. After five weekly doses she was discharged. She has been well up to the present time.

Case 6. Mrs. V. G. complained of "eye allergy" since the first part of July. Her eye lids had been very swollen, with severe itching since that time. She was told to omit the use of cosmetics. Skin tests on October 28 revealed she was positive to dust and molds, but negative to ragweed and weeds. She was treated with injections of dust and mold, plus staphylococcus toxoid, one unit at four-day intervals for seven doses, at which time her eye condition returned to normal. Her cosmetics were used then with no return of symptoms. She has had no recurrence.

Case 7: For three weeks Miss E.M. had complained of severe itching, burning and swelling of the right eyelids. She had had a similar attack in the left eye the previous August. The tree pollen count had been running very high (March 16) and this was the suspected allergen. Intradermal tests to tree pollen, as well as dust and molds, were strongly positive. One unit of staphylococcus toxoid was given. The eye improved and in a week the symptoms had almost subsided. She was dismissed completely recovered after two additional treatments with staphylococcus toxoid and dust, mold and tree pollen injections.

Case 8. Mr. C.H. was seen August 30 with the complaint that both eyes had been severely inflamed with intense itching and swelling of the lids. He had no nasal difficulties. An eye smear revealed four plus eosinophils. Since it was the ragweed season, this was the suspected trigger allergen. Tests revealed strong positives not only to ragweed but also to dust, molds, tobacco smoke and grasses. He was given one unit of staphylococcus toxoid. He returned in six days much improved. He was then given staphylococcus toxoid, 0.5 unit, with injections of dust, molds, ragweed, tobacco smoke and grasses. He returned in six days with complete relief of symptoms. The treatment doses with the toxoid were repeated with ragweed therapy the rest of the season without recurrence.

Case 9. Mrs. C.C. complained (November 28) of burning, itching, lacrimation, aching of the eyes, and a sandy feeling, with edema of the lids. These symptoms had been present constantly for two years but were slightly worse in the winter. There had been periods of less severity, but she had never been free of symptoms. She also gave a history of frequent colds, winter and summer but more severe in the latter. Examination revealed an edematous conjunctiva and skin of the lids. A nasal smear was packed with eosinophils in clumps. Allergy tests to dust, molds, ragweed, grasses and tobacco smoke were positive. She was given one unit of staphylococcus toxoid, and this was repeated every other day for four doses by her local physician. She reported that she was much worse the following day after her tests and remained so for seven days. She felt somewhat better upon her return to the office December 7. Treatment doses of the positive allergic inhalants were given and the staphylococcus toxoid reduced one half unit every third day. On December 13 she reported that her eyes had been much worse the day after treatment but improved during the week. The treatment dosage was reduced to avoid reactions and the staphylococcus toxoid continued on the same basis. Due to the nasal condition and the

extreme reaction to the inhalant treatment doses, it was suspected that a food allergy was present. She was given clinical food tests, as described by Rinkel.[3] It was found that her symptoms could be produced by eating Irish potatoes and/or chocolate candy. These foods were removed from the diet, and it was then found that she could be controlled easily by the optimum inhalant dosage once a week, plus staphylococcus toxoid, 0.5 unit. She has been comfortable and symptom-free since.

Case 10. Mr. W.L. reported for treatment October 27, 1950. He complained of itching, burning, redness and swelling of the lids for three weeks. Examination revealed a kerato-conjunctivitis and edema of the lids with dermatitis. Tests for dust, molds, ragweed and weeds and tobacco smoke were positive. He was given an injection of staphylococcus toxoid, one unit. On October 30 he reported that his eyes had been worse immediately after the tests but gradually improved through the week. Treatment doses of ragweed, dust, molds, and tobacco smoke were given once a week with a complete control of symptoms.

SUMMARY AND CONCLUSIONS

1. Seasonal allergens are very often the primary etiologic cause of allergic conjunctivitis and dermatitis of the lids.

2. Contact dermatitis of the lids may not result if the patient's allergic state is not accelerated by seasonal allergens.

3. All cases should be tested for seasonal allergens and treated specifically.

4. Staphylococcus toxoid is a good nonspecific treatment and can be used in conjunction with specific treatment.

5. Mere withdrawal of local contacts will, in many cases, give no results unless nonspecific and specific allergic treatment are given.

6. Foods may often be a complicating factor, and unless they are found and eliminated, good results will not be obtained.

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THE USE OF STAPHYLOCOCCUS TOXOID IN THE TREATMENT OF ALLERGIC PENICILLIN REACTIONS

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THE story of how penicillin's amazing antibacterial action was discovered in 1928 by the British bacteriologist, Alexander Fleming, is too well known for repetition here in any great detail. We might consider, however, the leading events in penicillin research and development.

In September 1928, Fleming[4] discovered the accidental contamination of an agar plate on which staphylococci were being grown by a greenish, bushlike mold belonging to the generic family Penicillium, which destroyed all the germs in its vicinity.

In 1929 Fleming announced its discovery, but for the next ten years, 1929 to 1939, general scientific and medical interest in Fleming's discovery was practically nil.

From 1939 to 1941, Florey, Chain, Heatley and associates working at Oxford University learned how to purify the drug, developed its pharmacology in animals, and established a good groundwork for penicillin therapy.[4]

In 1941, Florey and Heatley came to the United States and enlisted American interest in the production of enough penicillin for clinical studies in allied military needs.

In 1942, manufacturers in the United States were able to produce only enough penicillin to treat 100 patients. However, in 1943, production began to proceed rapidly, and from a few million units in January, it rose to five billion units in November 1943. This advanced to 70 billion units in April 1944, and 130 billion units in July 1944. By June 1945, production had been increased 2,500 times in two years. Production was further increased twenty-fold between 1945 and 1950, so cost of penicillin was markedly reduced.

More widespread use of the drug soon developed. Penicillin lozenges, tablets, injections, inhalations, drops, ointments, and penicillin combinations with other antibiotics all came into use.

With this widespread use of penicillin, a goodly number of patients began to manifest allergic reactions to the drug. Therefore, the indiscriminate use of penicillin for colds and simple infections is to be condemned.

As stated by Hansel,[2] the most common type of reaction manifested to penicillin simulates serum disease. It is characterized by the same incubation period of 7 to 10 days, followed by urticaria, angioneurotic edema, itching, fever, and joint pains. A considerable number of such cases may be controlled by the administration of the antihistaminic drugs, but the more severe reactions cannot be controlled by such ordinary methods.

Peck and his colleagues[5] have stated that there are two distinct types of reaction to penicillin: (1) the serum-sickness-like urticarial type, which is an induced sensitivity, and (2) the eczematoid-trichophytid-like type, which may be based upon a previous sensitivity produced by a fungus infection. The so-called "spontaneous" penicillin-sensitive cases belong to this latter group. Exfoliative dermatitis following peni-

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cillin administration appears to be of rare occurrence and of the mild type.

According to Hansel,[2] the face and mouth particularly appear to be more susceptible to penicillin; the aerosol resulting in stomatitis, nasal irritation and dermatitis around the nose and mouth in about 5 per cent of the cases, while stomatitis from oral administration occurs in about 14 per cent.

Keefer[3] has stated that penicillin by mouth causes fewer or no disagreeable side reactions, and that the incidence of hypersensitivity is less than when it is given intramuscularly.

Epstein and Macaulay[1] point out that if a reaction occurs during parenteral penicillin therapy, it does not necessarily indicate that penicillin may not be tolerated at a later date. Sensitivity may be of relatively short duration and may decline rapidly. It may decrease over a period of 6 to 12 months so that a second course may be taken without reaction. Each subsequent attack may tend to increase the degree of sensitivity with an increase also of the severity of the reaction.

Peck and his associates[5] performed tests for penicillin and trichophytid sensitivity in a group of 406 adults and 91 children. They used intradermal tests:

A. .01 to .02 cc. of 5,000 units per cubic centimeter of amorphous penicillin; read in 15 to 20 minutes for immediate reaction.

B. .10 cc. intradermal, 2,000 units in .10 cc. isotonic sodium chloride; delayed type of reaction is read in 48 hours or later.

C. Trichophytid tests: 1:30 dilution in .10 cc. of isotonic sodium chloride.

The delayed test, similar to the trichophytid test, is reliable for penicillin sensitivity. A positive reaction is characterized by an area of erythema with edema and infiltration about 1 cm. in diameter.

According to Peck,[5] two major types of penicillin reactions are most commonly encountered: (1) reactions of the urticarial serum-sickness-like type in which sensitization is induced by treatment; (2) reactions with an erythematovesicular eruption resembling the trichophytid.

REACTIONS OF THE URTICARIAL SERUM-SICKNESS TYPE

This constitutes the most common type of allergic reaction to penicillin and is characterized by urticaria and erythema with joint pains and sometimes fever. The incubation period is usually 7 to 12 days, but may occur in several days or may be delayed as long as three weeks. Severe cases may have angioneurotic edema or asthma. Most reactions follow intramuscular injection, but they may also follow oral administration. A negative skin reaction to penicillin test does not necessarily exclude sensitivity. More positive skin tests occur in those patients who have previously had penicillin. Peck observed that of 130 patients who received penicillin, 12 or 13.4 per cent showed positive cutaneous reactions. Among those that had not had penicillin, the incidence of reaction was 5 per cent. The incidence of penicillin reaction is very low in children.

REACTIONS WITH ERYTHEMATOVESICULAR ERUPTIONS, TRICHOPHYTID TYPE

An erythematovesicular reaction sometimes occurs. It tends to localize on the hands, feet and groin and may become a generalized exfoliative dermatitis. This may represent a secondary mold infection and not an allergic reaction.

When patients who have had mild penicillin reactions require further penicillin administration, antihistaminics may be used. Small trial doses of the penicillin may be given at six hour intervals and gradually increased. It is cer-

tainly unwise to give large doses to patients who report previous penicillin sensitivity.

Often mild reactions to penicillin disappear without any treatment, and slightly more marked reactions may readily respond to antihistaminics and epinephrine. However, severe reactions often do not respond to ordinary treatment, and more intensive types of therapy and control must be instituted.

Hansel[2] states that Rinkel found that satisfactory relief for acute urticarial reactions could be obtained by the administration of one to two units of penicillin intradermally, given every one or two days.

Prince and Etter[6] reported 8 cases of penicillin reactions favorably treated with administration of histamine intradermally and intravenously. The intravenous solution was .0275 mg. of histamine base in 250 cc. of saline, slowly injected so as to produce a generalized flush. It may be repeated in six to eight hours. The intradermal injections were small doses: .1 cc. of a 1:5000 dilution or .05 cc. of a 1:100 dilution.

At the suggestion of Hansel, we treated 48 patients who had penicillin reactions with small doses of staphylococcus toxoid. This was found to be very effective, often dramatically so in many cases in which other methods of treatment had failed. The mechanism appears to be one of a foreign protein type of reaction which breaks up the chain of allergic responses.

Treatments were originally started by the injection of one to two units of staphylococcus toxoid. These were given at 24- to 48-hour intervals and gradually increased to three, four or five units. Soon it was found, however, that the smaller dose, 0.5 unit, seemed to be much more effective in producing prompt relief of symptoms. Thus, the more recent cases were started on one unit, and then, in 24 to 48 hours, 0.5 unit, or even 0.25 unit, was given. Frequently, two or three injections were sufficient to create complete relief of symptoms. In many cases, only one dose was needed, and often relief was noted in a matter of hours. In a few cases, four or five injections were needed, but in most cases, the average to obtain complete relief was two or three injections of 0.5 unit, given at 48-hour intervals.

The preparation used was staphylococcus toxoid, dilution 1, 100 units per cubic centimeter. One cubic centimeter of this solution was diluted with 9 cc. of normal sterile saline, thus producing a solution containing 10 units per cubic centimeter. One unit dose is equal to .10 cc.

Of the 48 cases treated, the average time of onset of postpenicillin urticaria was 7 to 10 days. However, a few patients manifested reactions within 48 hours, and in an occasional one, as late as three weeks after injection. Eight patients presented severe reactions with swelling of hands and feet, edema of tongue and lips, and severe joint pains. In all instances, relief was obtained satisfactorily. Six patients hospitalized with general diffuse postpenicillin urticaria were promptly relieved where other methods of treatment had failed.

Several cases of chronic urticaria were treated with satisfactory improvement. Three cases of infantile eczema of six years duration were relieved by four or five injections of staphylococcus toxoid.

Thus, in conclusion, it seems that this method of treatment has given consistently effective relief, and its further use is suggested as a satisfactory method of control for such reactions.

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A SOCKET STRETCHER FOR DEFORMED SOCKETS

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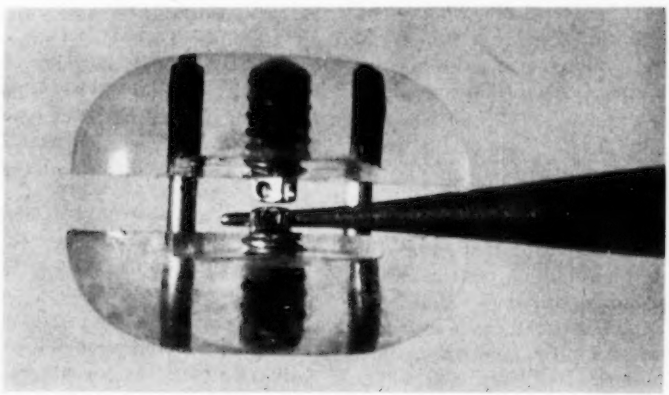

FIG. 1.—Stretcher for deformed sockets.

THERE are occasional cases of deformed anophthalmic sockets in which some means of stretching the socket and deepening the cul-de-sacs may eliminate further surgery.

This acrylic plastic socket stretcher* can be made in any size and works much like a shoe tree. After inserting the stretcher in the socket, it is easily widened by two central set screws. These set screws have square heads with holes in each side facilitating rotation of the screws by a small punctum dilator. These stretchers work well and can be tolerated in the socket indefinitely.

The small model depicted was made for a four-year-old child born with congenital anophthalmos and a very tiny rudimentary socket. Plastic surgery was done to make a palpebral aperture of normal width and a socket lined by a skin graft. Due to moderate contracture, an eye could not be fitted. After wearing this stretcher for five months (adjusted larger each month), the cul-de-sacs were deep enough to accept a prosthesis.

* Manufactured by Storz Instrument Co., 4570 Audubon Ave., St. Louis 10, Mo.

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HISTAMINE IN THE PRACTICE OF OTOLARYNGOLOGY

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CLINICAL FEATURES AND ETIOLOGY

HISTAMINE therapy in the field of otolaryngology has advanced rapidly since Horton presented his classical work on histamine cephalalgia, in which he used the intravenous technic. Hansel has further augmented the progress of this drug. Shambaugh, in a recent article, reported good results in postoperative fenestration cases, in which the hearing was failing, using dilutions of histamine up to 1:10,000,000 and higher.

We have followed Hansel's technic of high-dilution therapy in the treatment of hay fever cases, involving the ragweeds and other seasonable pollens, also in the use of house dust, staphylococcus toxoid and various allergens. This same high-dilution technic can be used in histamine therapy in dilution up to 1:10,000,000,000. All cases are titrated out from number 10 down to 9, 8, and 7, number 10 being 1:10 billion (numbers indicate number of zeros). A reading is taken after ten minutes; this is just a lead to detect the degree of reactability. The type of skin, room temperature, emotions, drugs, dilutions, etc., play a part in the type of reactions.

High dilution therapy has been used in headaches of various kinds, such as the histamine cephalalgias, the migraine headaches, the traumatic headaches following concussions, skull fractures, tension headaches, psychogenic headaches and vascular headaches. The pains are of two types in the majority of the cases, those which are due to involvement of the sensory nerve ganglion, causing neuralgia and pain, and those which are due to vascular tension, involving the sympathetic and parasympathetic nervous system. Histamine is also of value in (1) sudden loss of hearing whether on an allergic basis, traumatic basis, or following fenestration operations; (2) the neuralgias: trigenimal, glossopharyngeal, herpetic; (3) Ménière's syndrome complex, with its associated vertigo and tinnitus; (4) pains following trauma, concussion, skull fractures and cerebral hemorrhages; and (5) vasomotor rhinitis.

TREATMENT

A very careful history is taken on all cases, especially searching for allergic factors to be eliminated, as well as other pathologic conditions.

Allergy tests are done when indicated. A routine including avoidance or elimination of the offending allergen or desensitization to it is attempted.

If the case fails to respond, then histamine therapy is worth a trial. If histamine alone fails to elicit good results, then other allergic extracts, such as house dust and staphylococcus toxoid, are used along with drugs such as cafergone, dihydro-ergotomine tartrate (D H E 45), bellergal, the vasodilators such as roniacol, and the resins such as resodec when indicated, and any other form of therapy that may be beneficial. Histamine is effective synergistically with other drugs in many instances in which it is not effective alone.

On the basis of the history and the titrations, an index of the degree of sensitivity and the degree of response expected may be determined.

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These patients are routinely started on number 10 dilution (1:10 billion), 0.1 cc. subcutaneously, and are also put on sublingual drops, 1:10 thousand, 2 drops underneath the tongue, twice daily. The patient is asked to return in four or five days, when a second evaluation of the symptoms is made whether he is better or worse, and whether it is necessary to change the dosage level, eliminate different causative factors, etc. This high-dilution therapy of Hansel's has worked very well; for practical purposes it eliminates a great deal of skin testing if a very careful allergic history is taken. Histamine therapy should be tried before preparing a large series of skin tests or advising hospitalization. As a rule, three or four treatments will give the patient relief.

The optimum dosage level can usually be determined after the second or third injection. Once the patient has responded to a certain dosage, that dosage is not changed as long as good results are obtained. This is an important point. The effective dosage should be maintained. The time interval may be changed gradually from twice a week to once a week, then to ten days, two weeks and three weeks. Then treatment may be discontinued to determine how long the patient remains symptom free. Some patients will become free of symptoms after four or five treatments, many within two or three weeks. Some may have to continue treatment for many months. Many patients recover, remain well for several months or years, then have a recurrence. After two or three injections, the symptoms may again subside. The patient learns to evaluate his symptoms and returns when in need of further treatment.

Case 1. R. E. W., white, male, age 60, in the insurance business, had a diagnosis of histamine cephalalgia.

The patient has severe attacks, waking him up at night, making it necessary to get up and walk. He had severe pain in his right eye, congestion in the right side of his nose and tenderness over the side of the face. He also had an associated anginal attack of pain. He was placed on 0.1 cc. of number 10 histamine, twice a week for three weeks. The pain stopped completely as well as the anginal attacks. After the discontinuation of treatment, he had a recurrence and was again treated for two weeks. The symptoms subsided the second time and there has been no recurrence for the past year.

Case 2. N. W., white, male, age 37, personnel director for a large manufacturing concern, also had a diagnosis of histamine cephalalgia.

The patient complained of severe headaches on right side, waking him up in the night. The pain was localized in the eye, often causing the eye to tear. He also had congestion of the nose lasting about an hour. The attack was so severe that he had to get up and walk. He also had difficulty in raising the lid. He had no previous history of headaches and there was no history of allergy. Treatment was started with 0.1 cc. of number 10 histamine, and injections were given twice a week, with excellent results. He was treated for just one month and has never had a recurrence.

Case 3. P. W. B., white, male, age 42, a telegrapher.

The patient complained of facial neuralgia on the right side; shaving or rubbing a washcloth on the face would start terrific pains in that region. He was started on 0.1 cc. of number 10 histamine, which resulted in a remission of symptoms. This occurred two years ago. He was treated for two months; pains ceased and he remained free of symptoms for a period of six months. When a slight recurrence appears, he returns for two or three injections, which give him relief for a period of months. This patient can anticipate when attacks are expected. Two or three injections usually prevent severe attacks. He has followed this course for the past two years. The advantage of this therapy is that patients do not have to be hospitalized. They can carry on their occupations and quite frequently obtain complete relief, in a comparatively short time.

Case 4. Mrs. C. S., white, female, age 38, housewife, had a diagnosis of migraine.

History of migraine for a period of fifteen years. The attacks were very severe, requiring her to go to bed with scintillating

scotoma, blindness, nausea and vomiting. She has been treated with histamine, 0.1 cc. of 1:1,000,000 since February 17, 1950, and has been very comfortable but cannot go without therapy.

Case 5. Mrs. E. D., white, female, age 62, had a diagnosis of Ménière's syndrome. The patient complained of extreme dizziness, ringing in the ears and impaired hearing. She was placed on 0.2 cc. of number 10 histamine twice a week for one month, and symptoms have cleared up completely.

Case 6. Mrs. W. B., white, female, age 84, had a diagnosis of cerebral hemorrhage. This patient was unable to speak, had difficulty in opening her mouth, and was unable to push her tongue forward. She was given histamine therapy, 0.1 cc. of number 8, and within ten minutes after she was given the intradermal injection she was able to open her mouth, move her neck freely and swallow. She was treated for a period of three months, after which she moved from town. She derived a great deal of benefit from the intradermal injections. Because she could not speak, she always brought a note describing her condition. This is a sample:

"I was so good after the last treatment, could swallow much better, missing last week, the past three days I can't swallow so well and neck pains but does not fall forward so bad. Can lie down on one pillow, sleep better."

COMMENTS

These patients can be treated in the office, and after the maintenance dose has been established, very little time is required of either the patient or the physician. The patient soon works out the time intervals for treatment. This technic avoids hospitalization, loss of time and expense. Reactions such as falling blood pressure, thrombosis, headache, urticaria, asthma, tachycardia, hyperacidity, and gastric ulcers are complications rarely encountered when using high dilutions. Gastric analysis and alkaline therapy are unnecessary. This technic is not a cure-all but is offered as a suggestion for trial in the office before the expense of hospitalization is advised.

SUMMARY

The clinical features and possible pathogenesis of headache, Ménière's syndrome, deafness, neuralgia, posttraumatic and postoperative pain are discussed, and a course of high-dilution therapy, following Hansel's technic, is described.

High-dilution therapy, intradermally and subcutaneously with sublingual medication twice daily, is offered for your approval with the hope that it will be tried first, or after other forms of therapy have failed, as this method eliminates the more formidable technics and expense, time loss, and hospitalization.

House dust therapy, pathogenic molds, staphylococcus toxoid, vasodilators, resins, and other forms of therapy often have a synergistic action with histamine and aid a great deal in cases which resist specific histamine therapy.

HISTAMINE IN BELL'S PALSY

D. A. SKINNER, M.D.
NEWARK, OHIO

BELL'S palsy, commonly called rheumatic facial paralysis or refrigeration palsy, is a peripheral paralysis of the facial nerve due to a primary vasospasm of the stylomastoid artery, its branches and anastomoses. In peripheral facial paralyses due to infection, trauma, neoplasm, or hemorrhage, the vasospasm is secondary. Such cases are not termed Bell's palsy. The terms "primary and secondary vasospasm" are used to differentiate those of presumed idiopathic origin from those of known origin.

The question concerning the basic and precipitating causes of the pitfalls of the vascular system is a complex one. There are many and various elements that work in unison to create this vasospastic pattern. Undoubtedly, heredity, vitamin and mineral deficiency, constitutional factors (especially those causing anemia), trauma, stress and strain, and glandular dysfunction help to create a reactive state so favorable that an imbalance of the autonomic system may occur.[11] With the framework of the autonomic system at the breaking point, it is probable that food, inhalant, physical, and psychosomatic allergies are the precipitating factors. The precipitating factors may also be basic ones.

The onset of many of these vascular disturbances occurs in the Spring and Fall seasons. It is common knowledge that many patients become more susceptible to foods during the pollen and dust seasons. Sudden changes of weather also play a part. Frequent repetitions of the same food may create new allergies. Tolerance[7] is often regained within a period of time by the omission or proper spacing of the offender. Food and inhalant allergens have a wide range of specificity. For instance, one may create a vertigo, another urticaria, and still another, a migraine.

The presence of tree and grass pollens in the Spring and of ragweed pollen and dust in the Fall may be the etiologic factor causing the seasonal increase of these vascular disturbances. This may be the explanation of many cases that have not been precipitated by exposure to cold, heat, or emotional upsets. However, inhalants and foods may have helped to promote the autonomic imbalance.

Approximately 70 per cent of the cases of Bell's palsy are precipitated by exposure to cold. A small percentage of cases follow exposure to heat and emotional upsets. Vestibular testing with cold water, the use of the ice collar, and dental extractions have been thought to be precipitating factors in rare cases. Pollen, dust, and foods probably play more of a part in the precipitation of these vascular phenomena than we have previously realized.

In a bonelike cavity, canal, or foramen, where important structures are enclosed, a serious hazard may arise when vasospasm[4] begins. This is also true in many other otolaryngologic conditions. Ménière's disease presents a typical example. Many authors of textbooks and articles have overestimated the importance of infection in many vascular disturbances, but they have said little or nothing about vasospasm. However, vasospasm secondary to infection must not be overlooked.

Presented at the meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, Oct. 19, 1951, Chicago, Ill.

The symptomatology of Bell's palsy is well known. The close proximity of the contents of the facial canal to the external auditory meatus probably accounts for the abrupt onset of paralysis in some individuals after exposure to cold. Other nerves of the body are not so affected by exposure to cold, due to the fact that they are better protected by soft parts and have no close proximity to the exterior. Females appear to be more susceptible than males and recurrences are not uncommon. More left facial nerves are affected than right. Rather large external auditory meatuses have been observed in most cases where refrigeration was a factor. No age group is exempt. The incidence is more common in the middle-aged group.

The site of the lesion of the peripheral part of the facial nerve can be localized at different levels by the presence or absence of taste and tears.[10]

Inasmuch as Bell's palsy is due to vasospasm, therapy must be directed toward vasodilatation or breaking the block. Histamine diphosphate is one of our best vasodilators. Loomis[6] administered histamine by the intravenous method to 5 cases with good results. In our hands, histamine diphosphate given subcutaneously by the low-dose optimum method, as advocated by Hansel,[2] is easily administered and has given excellent results.

Histamine diphosphate given by the low-dose optimum method is usually started with 0.1 cc. of the 10,000,000 dilution and gradually increased until the optimum effective dosage has been reached. These injections are given subcutaneously every two or three days. When improvement occurs, the patient is kept on that amount with very little change.

The paralysis recurred in several patients when dosage was increased above the optimum effective amount.[9] However, restoration takes place when the proper effective dosage is again repeated.

Proof of the vasodilating character of histamine diphosphate given subcutaneously by the low-dose optimum method can be demonstrated by the flicker photometer.[5] Vasodilatation, as shown by this test, is often sustained for a period of fifteen to thirty minutes. Controls of normal saline were used. The excessive administration of this vasodilator may lead to vasospasm.

Case S., white, male, age 53, was first observed on June 4, for Bell's palsy of the left side. The onset of paralysis occurred suddenly after sitting in a draft on May 12. His past history was negative except for migraine headaches and occasional attack of Ménière's symptom complex. Physical examination was negative except for a Bell's palsy of the infrachordal type.

Histamine diphosphate therapy was started by the low-dose optimum method. The initial dose was 0.1 cc. of the 10,000,000 dilution. Injections were given every two or three days with gradually increasing doses. After five weeks of therapy, there was little or no improvement. At this time, an attack of vertigo developed which incapacitated the patient. A few deliberate food tests were made and, after the ingestion of milk, a severe vertigo was created. Flicker photometer testing demonstrated that milk had increased the vasospasm. This same testing also showed that the 10,000,000 dilution of histamine diphosphate was creating additional vasospasm. We decreased the histamine dosage to a vasodilating one of the 100,000,000 dilution and eliminated milk from his diet. Within a period of a week, his Bell's palsy had improved 75 per cent and his vertigo had completely subsided.

Summary

Bell's palsy is due to vasospasm.

The basic and precipitating causes may be interwoven with no definite line of demarcation.

Inhalants and foods must not be overlooked as basic and/or precipitating factors.

Therapy must be directed toward vasodilatation or breaking the block.

Many vasodilators, sympathetic paralyzing drugs, and glandular extracts may be interchangeably used with good results.

In our hands, histamine diphosphate, given subcutaneously by the low-dose optimum method as advocated by Hansel, is easily administered and has given phenomenal results.

Flicker photometer testing is an aid in determining whether a food or a given drug leads to vasospasm.

Proof of the vasodilating character of histamine diphosphate given subcutaneously by the low-dose optimum method can be demonstrated.

About 80 per cent of the cases of Bell's palsy give a phenomenal response to histamine diphosphate administered by the low-dose optimum method. The remaining 20 per cent usually give a partial response. Rarely is there no improvement.

HISTAMINE IN OPHTHALMOLOGY

The use of histamine in ophthalmology is becoming more popular. This is probably due to a better knowledge of the eye and especially to a better understanding of the pitfalls of the vascular system in and around the eye. Many of these pitfalls are due to the vasospastic cycle. In most ways, these idiopathic vascular disturbances of the eye resemble those of Bell's palsy. The globe, apex of the orbital cavity, optic foramen, and orbital fissures constitute suitable spaces that become hazards when vasospasm develops.

In the past, episcleritis has been thought to be due to infection. Focal infections were sought for and often treated or eliminated. However, the treatment or elimination of these infections did not always bring relief.

Duggan[1] suggests that episcleritis is of vascular origin and is a manifestation of acute localized tissue anoxia caused by vasospasm.

The basic and precipitating etiology of episcleritis appears to be similar to that of Bell's palsy. Undoubtedly, many cases are precipitated by exposure to cold.

Therapy must be directed toward vasodilatation. Histamine is given subcutaneously by the low-dose optimum method. Histamine diphosphate in dilutions from 1,000,000 to 1,000 is instilled in the eye every two or three hours. Care must be exercised in using the stronger dilutions as they may produce edema of the conjunctiva. This rarely occurs, but if it does, adrenalin, 1:1,000, will counteract the reaction. Some authors[8] have reported good results with local histamine therapy without additional treatment.

In the past, the etiology of retrobulbar neuritis was thought to be due to an inflammation of the optic nerve behind the eyeball.

Undoubtedly, a great many of these former cases that were thought to be due to infectious processes were the result of those factors that create vascular disturbances. However, a small percentage may be the result of a secondary vasospasm from focal infections, especially of the sinuses. Many authors state that retrobulbar neuritis is the forerunner of multiple sclerosis, as it is an early symptom in over half of the cases.

Therapy is directed toward vasodilatation. About 80 per cent give a response to histamine diphosphate administered by the low-dose optimum method.

Case: Since 1942, a male patient in his early thirties has been under my care for frequent attacks of retrobulbar neuritis. Intravenous typhoid was used on several occasions with good results. Low dose histamine was later used, starting with 0.1 cc. of the 10,000,000 dilution and increasing 0.1 cc. every two or three days until the optimum effective dosage was reached. After two or three injections, there was a marked improvement of vision and fields. In 1948, he was skin-tested and found

sensitive to cat hair. After the elimination of four cats from his home and hyposensitization for cat hair, there have been no recurrences to date of retrobulbar neuritis.

Sudden, complete, or partial paralysis of the extraocular muscles is not too uncommon in practice. Paralysis of the external rectus is the most common. The muscles supplied by the third and fourth nerves are about equally affected. Unquestionably, the etiologic basis for this condition is vasospasm.[3] Some cases may result from a secondary vasospasm due to infection but most arise from the primary.

In a summary of 18 cases, 5 were of the third nerve, 2 of the fourth, and 11 of the sixth. History, physical, and laboratory tests were negative, except for the following: One case involving the third nerve had syphilis, 2 involving the sixth nerve had diabetes, and another of the sixth nerve was due to trauma. All 18 were treated with histamine therapy. Fifteen responded, 5 received no benefit, 2 were cured after one injection, and 3 relapsed but later improved.

Summary

Many common vascular disturbances of the eye are the result of a primary vasospasm.

The basic and precipitating causes of most vascular phenomena of the eye are similar to those of Bell's palsy.

Therapy must be directed toward vasodilatation or breaking the block.

In our hands, histamine diphosphate, given subcutaneously by the low-dose optimum method as advocated by Hansel, is easily administered and has given phenomenal results.

Cortisone and ACTH are gaining popularity as their uses become better defined.

Foods that create vasospasm must be eliminated.

Inhalant allergies that create vasospasm must be treated.

Beware of the excessive administration of histamine diphosphate, especially the dosage that creates vasospasm.

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TRANSACTIONS
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All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

ASSOCIATION GROUP INSURANCE

The Council, by unanimous vote, has approved the Continental plan of Association group insurance for Fellows of the Academy designed by the Continental Casualty Company Associated Group Division. The plan proposed for the Fellows of the Academy is somewhat similar but not identical with that accepted by the American College of Surgeons. They are proposed by the same insurance company.

The members of the Council were made acquainted with the details of the plan, and many of them made comprehensive inquiries among experts in the type of insurance proposed. By mail vote, they authorized adoption of the plan by the Academy. The application for the plan was signed by President James Milton Robb and forwarded to the representative of the insurance company in Chicago. The plan will be put into effect as soon as a sufficient number of applications from the Fellows have been received. An invitation to join the plan will be mailed to all members of the Academy within a few days.

This plan is put into effect at no expense to the Academy and the Academy is in no way obligated to carry out any of its provisions. After careful consideration of the provisions proposed by the Continental Casualty Company in comparison with similar plans endorsed by a large number of professional groups, the Council has confidence that the insurance provided under the terms of this plan will inure to the credit of all members of the Academy.

WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

April 23, 1952

INSTRUCTIONAL COURSES IN OTOLARYNGOLOGY

THIS year we are attempting to stimulate more interest in the instructional courses in Otolaryngology and Maxillofacial Surgery. There are 127 courses in all, 103 individual and 24 continuous. The basic subjects are excellent for the individual who has been in practice for some time because they serve as refresher courses. The instructors have spent many hours in the preparation of their subjects and the courses are worthy of full attendance. We urge that more individuals attend these courses because they offer both direct and indirect value in the practice of otolaryngology and maxillofacial surgery.

We are inaugurating a new type of course this year—a question and answer discussion period. The panels are designed so that the practitioner may bring up a question regarding basic subjects as well as medical and surgical problems relating to the ear, sinuses, larynx, bronchi, and esophagus; the use of drugs in the practice of otolaryngology, and facial pains and headaches. Each period will be conducted by a group of three men who are teachers in their respective fields. We would suggest that the members wishing to attend these courses procure their tickets by mail before the meeting and come prepared with a list of questions for the panel.

In conclusion we would like to make the following suggestions: (1) If possible, obtain your tickets for the courses by mail previous to the meeting. This eliminates congestion and waiting at the time of the meeting. It also assures one a better opportunity of obtaining the courses desired. (2) Attend some of the basic courses because they have a great deal to offer from the practical viewpoint. (3) The younger teachers deserve a trial. Register for and attend some of their courses. (4) Send constructive criticisms to the Secretary for Instruction in Otolaryngology and Maxillofacial Surgery regarding the various courses you have attended. This will be of value to the instructor. (5) Write to the Secretary immediately after the meeting suggesting new courses and instructors. We earnestly desire to give you the best programs possible but we need your assistance.

DEAN M. LIERLE, M.D.

TINTED OPTICAL MEDIA

IN the last year and a half the public has had offered to it any number of types of "tinted" glasses designed for various and sundry purposes. The public also has been bombarded with articles in lay, semiprofessional and professional magazines and journals, as to the pro's and con's of the propriety and authenticity of the use of tinted lenses. These articles and reports coming from good, bad and indifferent sources, are frequently contradictory in whole or in part, leaving the public, and often professional people, completely confused.

In view of this situation and in view of the highly technical nature of the subject, the Joint Committee of Industrial Ophthalmology representing the American Academy of Ophthalmology and Otolaryngology and the American Medical Association, was requested to set up a Sub-Committee to study the problem of "tinted optical media." This Sub-Committee, authorities on filter lenses and "tinted optical media," was instructed to clarify the basic principles involved, by definition, and to answer certain specific questions.

REPORT OF SUB-COMMITTEE

1. *Categories*

In the consideration of any discussion of "tinted optical media" (i.e., lenses, plano or prescription, made up into glasses and carrying a tint) it is obvious that these fall into several categories.

A. Sunglasses:
   1. Neutral
   2. Colored
   3. Polarizing
   4. Reflecting

B. Tinted glasses for constant wear.

C. Colored glasses for industrial, military or other specialized purposes. This category is not discussed.

2. *Definitions*

It is important to have clear definitions of these categories in mind during this discussion.

A. Sunglasses are lenses designed primarily for wear in sunlight of high intensity.

1. Neutral lenses absorb all wave lengths of the visible spectrum (light) in approximately equal degree. The light transmitted by such lenses is reduced in intensity but its color is essentially unchanged.
2. Colored lenses absorb the light rays of the various wave lengths in unequal degree. Light transmitted through such lenses is reduced in intensity and its color is modified by the unequal absorption.
3. Polarizing lenses absorb light rays which vibrate in some planes. Light vibrating in other planes is transmitted.
4. Reflecting lenses depend on a thin metallic coating which reflects a portion of the light. The intensity of light reaching the eye is thereby reduced.

The principles of absorption, polarizing, and reflection can be combined in a single lens.

B. Tinted glasses for constant wear are mildly colored lenses marketed for use in optical prescriptions. In general, these lenses transmit a high percentage of light.

Questions and Answers

A number of questions were given the Sub-Committee with reference to "tinted optical media"; these are presented exactly as given.

Q. *Does the wearing of tinted lenses increase visual acuity by reducing chromatic aberration or by eliminating scattered violet, blue, and green light?*

A. This Sub-Committee does not believe that acuity can be appreciably improved by the wearing of any tinted lenses and is not aware of any accepted study which supports such a thesis.

Q. *Do sunglasses diminish ocular discomfort?*

A. Sunlight of even high intensity will produce no discomfort in some people. Where discomfort exists, it is frequently diminished by wearing sunglasses.

Q. *Do sunglasses affect the perception of color?*

A. Colored sunglasses (in contradistinction to neutral sunglasses) affect perception of color to some extent. Under ordinary circumstances the distortion of color perception by the color-normal person is not significant. However, at least 5 per cent of our population is not color normal. The use of colored lenses by these people may exaggerate their defects and pose a serious problem. Even color-normal individuals frequently encounter situations in which colored glasses may constitute a hazard. (Example: The recognition of colored traffic signals viewed against the bright background of a sunset.)

Q. *Are there spectacles which absorb the wave lengths responsible for cataract?*

A. There is no conclusive evidence that daylight in any intensity, or over any period of time, produces a cataract.

Q. *Objections to tinted glasses arise when they are promoted with unfounded advertising claims. What claims can be permitted?*

A. The primary function of a sunglass is to reduce brightness. Therefore, the proper and principal claim that can be made for a sunglass is that it reduces glare in proportion to the density of glass. Additional descriptive claims may be made, such as: that the glasses are cosmetically pleasing, that they reduce ultraviolet and/or infra-red radiation, that they are essentially neutral, or that they have certain physical properties of shape, size, polish, base curvature,

or color. However, advertising statements must not imply that any of these characteristics are of physiologic benefit. We (the Sub-Committee and Joint Committee) cannot anticipate the multiplicity of claims that may be made and, therefore, every type of claim must be considered individually. The common practice of distorting scientific information for promotional and sales purposes is to be deplored.

Q. *Will you give a list of minimum requirements for the acceptability of sunglasses.*

A. Recognizing that inexpensive sunglasses have a definite usefulness, this Sub-Committee believes it unwise to establish requirements which might eliminate these glasses from the market. However, to inform and protect the purchaser, sunglass manufacturers should be encouraged to *label* their products with respect to the following properties:

1. Transmission of visible light expressed in percentage, together with the tolerance of deviation from the standard. (Example: Transmission 20% = 1%.)
2. Type of manufacture. (Example: "Ground and Polished," "Bentplate," "Blown," etc.)
3. Base curve. (Example: "6 Base Curve.")
4. Optical tolerances:
   a. Power (as worn)—stated in fraction of a diopter.
   b. Prism (as worn) — stated in fraction of a diopter.
   (Example: "Tolerance": Power—1/16: Prism 1/16 A.)
5. An expression of the percentage neutrality when a standard acceptable to the ophthalmic professions is developed.

General Comments

In considering the use of glasses to reduce overhead brightness either indoors or out, it is to be remembered that a visor or broad-brimmed hat is a most effective shield and will frequently obviate the necessity for sunglasses.

This Sub-Committee condemns the use of any type of "night-driving lens." Any such lens, whether colored, reflecting or polarizing, reduces the total light transmitted to the eye and renders the task of seeing at night more difficult. Similarly, it condemns the use of colored windshields (which may prove a hazard at night) or the promotion of removable filtering or polarizing shields as a useful aid in night driving.

As applied to the general civilian population, this Sub-Committee endorses the thesis that sunglasses should be worn only when the intensity of light produces discomfort, and that the penalty for their wear otherwise is the reduction of the individual's tolerance to bright light. Unless indicated by definite ocular pathologic processes, the habitual use of sunglasses indoors is most objectionable.

It is emphasized that no commercially marketed sunglass is sufficiently dense to permit direct gaze at the sun. Much denser filters are required for a safe view of a solar eclipse.

With respect to the mildly tinted lenses designed for constant wear, it is recognized that the more densely tinted lenses of this group are useful in the presence of photophobia due to pathologic conditions. In the absence of pathology those denser tints should not be prescribed for constant wear. The total light transmission of the lighter tints differs so little from that of crown glass that they are not considered effective filters in pathologic states. The Sub-Committee has no objection to the use of the very mildly tinted lenses if the patient desires them and can afford the additional cost. It is emphasized, however, that we do not agree that they offer physiologic advantage over crown

glass for use under fluorescent lighting or other lighting situations.

The Sub-Committee is aware of the investigations which have shown that prolonged exposure to bright sunlight impairs night vision subsequently. This factor is of extreme importance in certain military situations but of very questionable importance in civilian life. The motorist viewing the road ahead under headlights is dependent upon photopic vision; and we are not convinced that photopic vision is appreciably improved by the use of sunglasses. Rather, we believe that the inadvertent use of sunglasses at dusk constitutes a far greater hazard in driving on the highways than any possible decrement in vision at low levels of illumination.

The Sub-Committee agrees in general with the statements contained in the article by Dean Farnsworth, "Standards for Sunglasses," published in Sight Saving Review, 20:81-87 (Summer) 1950.

SUB-COMMITTEE

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LT. COMDR. DEAN FARNSWORTH, USN
EVERETT V. KINSEY, PH.D.
COL. VICTOR A. BYRNES (MC) USAF
(representing the Joint Committee)

In Memoriam

Harold Bailey.....Charles City, Iowa
March 9, 1952

Seth Arthur Brumm.....Philadelphia, Pennsylvania
February 21, 1952

George Hageman.....Longmont, Colorado
January 14, 1952

Hans Lauber.....Wien, Austria
March 3, 1952

George Guerin Marshall.....Rutland, Vermont
April 1, 1952

Walter Mudie Paton.....Vancouver, B. C., Canada
December 18, 1951

Malcolm Chester Pfunder.....Minneapolis, Minnesota
January 23, 1952

Roy Otis Riser.....Park Ridge, Illinois
January 3, 1952

Delano Richard Ryder.....Fall River, Massachusetts
December 19, 1951

William Walter Sauer.....Marietta, Ohio
June 30, 1951

Otto V. Mackenty Schmidt.....New York, New York
January 15, 1952

George Haeberle Stine.....Colorado Springs, Colorado
April 9, 1952

Karl Ludwig Stoll.....Cincinnati, Ohio
December 17, 1951

Henry DeWitt Watson.....Binghamton, New York
June 20, 1950

HOME STUDY COURSES

Application blanks for the 1952-1953 Home Study Courses may be secured from the Executive Secretary-Treasurer of the Academy, Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minnesota. Registrations must be completed before August 15. The fee is $20.00.

Dr. Lawrence R. Boies is Secretary for Home Study Courses and Dr. Hendrie W. Grant is his associate in charge of ophthalmology. The subjects covered and the present faculties are listed below:

OPHTHALMOLOGY		OTOLARYNGOLOGY	
September EMBRYOLOGY, ANATOMY AND NEURO-ANATOMY	Raymond E. Meek John W. Henderson *C. Wilbur Rucker	*September* ANATOMY I and *October* ANATOMY II	Maurice F. Snitman J. Brown Farrior *O. E. Van Alyea
October PHYSIOLOGY	Hendrie W. Grant George P. Guibor	*November* HISTOLOGY	Leland G. Hunnicutt Frederic J. Pollock
November OPTICS AND PRINCIPLES OF BIO-MICROSCOPY	Arthur Linksz Edwin F. Tait *Frederick C. Cordes	*December* PATHOLOGY	Herman Semenov Werner Mueller *L. W. Dean, Jr.
December REFRACTION	Daniel Snydacker Frank W. Newell *Thomas D. Allen	*January* PHYSIOLOGY	Anderson C. Hilding William C. Thornell *Carl H. McCaskey
January EXTERNAL DISEASES, BACTERIOLOGY AND VIROLOGY	James H. Allen S. Rodman Irvine Samuel N. Key, Jr.	*February* BACTERIOLOGY	Ben H. Senturia Henry L. Williams *Jack E. Mulsow
February PERIMETRY AND NEURO-OPHTHALMOLOGY	P. J. Leinfelder Cecil W. Lepard	*March* PHARMACOLOGY	Robert E. Priest Clifford F. Lake
March EXTRAOCULAR MUSCLES	Paul T. McAlpine Hunter H. Romaine *Conrad Berens	*April* HEARING FUNCTION AND TESTING	George E. Shambaugh, Jr. Arthur L. Juers *Theodore E. Walsh
April OPHTHALMOSCOPY AND MEDICAL OPHTHALMOLOGY	John P. Macnie Wm. Banks Anderson Samuel D. Aiken	*May* VESTIBULAR FUNCTION AND TESTING	William J. McNally John R. Lindsay *H. C. Ballenger *Moses H. Lurie
May PATHOLOGY	Michael J. Hogan John S. McGavic *Georgiana D. Theobald	*June* OTOLOGICAL ACOUSTICS	Scott N. Reger Clair M. Kos
June PHARMACOLOGY AND THERAPEUTICS	Kenneth C. Swan Alfred J. Kreft		

*Consultant

SECTION ON INSTRUCTION

* * *

FIFTY-SEVENTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 12-17, 1952
PALMER HOUSE
Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

This Section presents for 1952:

Ophthalmology: 107 Individual Courses, including 10 home study course discussion periods and one special course; 43 Continuous Courses. The faculty is comprised of 192 instructors, and the hours of instruction total 402.

Otolaryngology: 103 Individual Courses, including 6 round table discussion periods; 24 Continuous Courses. The faculty is comprised of 132 instructors, and the total number of instruction hours is 275.

General Instructions

Only members who have paid their 1952 dues may order instruction tickets in advance of the meeting. Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 10.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to W. L. Benedict, M.D., c/o Palmer House, Chicago, Illinois. Advance orders will not be filled if received after October 9.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two or three different days allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of

a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third preferences for each period in case first preference is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgement of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

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CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE 1

Room 738 Periods M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5

JAMES H. ALLEN, M.D., et al

Bacteriology and Infectious Diseases of the Eye

An eight-period course—$16.00

The first hour of the course will be devoted to bacteriology and methods of etiologic diagnosis of infectious diseases. Six hours will be devoted to the clinical manifestations and differential diagnosis of infectious and allergic diseases of the eye, and one hour will be devoted to a general summary of the therapy of infectious diseases.

M-4—Bacteriology
James H. Allen, M.D.
New Orleans, La.

M-5—Infections of the Lids and Lacrimal Apparatus
Hugh Ormsby, M.D.
Toronto, Ont., Canada
BY INVITATION

T-4—Bacterial Infections of the Conjunctiva
Isadore Givner, M.D.
New York, N. Y.

T-5—Virus Infections of the Lids and Conjunctiva
Phillips Thygeson, M.D.
San Jose, Calif.

W-4—Infectious Diseases of the Cornea
Trygve Gundersen, M.D.
Boston, Mass.

W-5—Allergic Diseases of the Lids, Conjunctiva, and Cornea
Frederick H. Theodore, M.D.
New York, N. Y.

Th-4—Infections of the Orbit and Globe (to include surgical infections)
Henry Allen, M.D.
Boston, Mass.
BY INVITATION

Th-5—Therapy of Infectious Diseases
Alson E. Braley, M.D.
Iowa City, Iowa

COURSE 2

Room 713 Periods W-5, W-6

KARL W. ASCHER, M.D.
Cincinnati, Ohio

Aqueous Veins in Glaucoma

A two-period course—$4.00

In addition to fluid exchange through capillaries, the canal of Schlemm and the aqueous veins provide for elimination of intraocular fluid. Originating in, or near the limbus, the aqueous veins form, together with the canal, a physiologic unit of common origin and of clinical interest. Recent histologic investigations have confirmed the original assumption that the aqueous veins originate directly from outlets of the canal of Schlemm.

Mathematical evaluation of anatomic data elucidate the pressure gradients and fluid output of normal and of glaucomatous eyes. In both types, aqueous veins are found; less readily, however, in eyes with inveterate and with secondary glaucoma due to iridocyclitis. In eyes suffering from hydrophthalmos and buphthalmos they are rarely found.

Intravascular pressure in aqueous veins of glaucomatous eyes seems to be lower than in aqueous veins of normal-pressure eyes; diurnal oscillations seem to be present, at least in some glaucomatous eyes. Premonitory signs of decompensation can be detected in aqueous veins. Pulsations seem to be less frequent in aqueous veins of glaucomatous eyes than of eyes with normal pressure. Miotics, privine and neosynephrine, increase the clear fluid content of aqueous veins in normal-pressure and in glaucomatous eyes. After successful surgery, formerly invisible aqueous veins may appear; postoperative hypotony, however, may efface previously visible aqueous veins. The result of compression tests is different in normal-pressure eyes than in glaucomatous eyes, whether pressure is exerted on the recipient vessel of an aqueous vein or on the cornea, or on the sclera. Attempts have been made to evolve a clinically valuable glaucoma test on aqueous veins.

Study of the aqueous veins of glaucomatous eyes seems to support the retention theory of glaucoma which is given further

credence by recent investigations of the resistance to outflow under tonometer pressure.

COURSE 3

Room 724 Periods M-5, T-5, W-5, Th-5

ARTHUR J. BEDELL, M.D.
Albany, N. Y.

Medical Ophthalmoscopy

A four-period course—$8.00

A review of the common and rare fundus lesions. A demonstration of more than 600 kodachrome fundus photographs. Hypertension, diabetes, nephritis, tuberculosis, syphilis, the blood states, increased orbital and intracranial pressure as evidenced by papilledema, optic neuritis of various types, and the optic atrophies will be presented and discussed.

COURSE 4

Room 724 Periods M-4, T-4, W-4, Th-4

MILTON BERLINER, M.D.
New York, N. Y.

Advanced Technics in Slit Lamp Microscopy

A four-period course—$8.00

The purpose of this course is to review the different methods of illumination and to show their practical application in the observation of normal and pathologic findings. To be considered among these are scatter, direct focal, retroillumination, and specular reflection. A rapid method for the systematic examination of the eye by these methods is explained. Appliances and technics for the biomicroscopic examination of the deeper vitreous and fundus will be demonstrated and discussed. Also to be stressed is the importance of examination of preoperative and postoperative cases by these methods. Numerous lantern slides will be used to illustrate special clinical entities. Newer conceptions of lens changes (cataracts) and vitreous alterations will be presented.

COURSE 5

Room 781 Periods Th-4, Th-5

HENRY L. BIRGE, M.D.
Hartford, Conn.

F. L. PHILIP KOCH, M.D.
Bronxville, N. Y.
and
CHARLES BARBER, M.D.
Hartford, Conn.
BY INVITATION

New Theories of Retinal Venous Thrombosis

A two-period course—$1.00

Retinal venous thrombosis occurring in patients beyond middle life may often be attributed to an excess of red blood cells or a mild form of polycythemia. We have been checking the blood volume of our cases and find that low grades of polycythemia are frequently associated with retinal venous thrombosis. Blood volume examinations are detailed laboratory procedures in which the ratios of the various blood components are estimated accurately.

It is logical to suppose that a retardation of the blood flow through the smaller vessels of the retina may predispose to thrombosis. In cases in which only one branch of the central retinal vein is affected, the outcome has been satisfactory in that the vision has returned to normal following a reduction of the polycythemia to normal. When massive thrombosis takes place in the retina, the outcome is never satisfactory. Complications such as diabetes adversely affect the outcome.

Cases in which volume studies should be performed are those in which the retinal veins are enlarged, engorged and slightly cyanotic. The practice of checking blood volume preoperatively in patients who may be liable to thrombosis in other areas besides the retina may prevent complications in ocular surgery.

A fact of striking significance in polycythemia is that 56 per cent of the deaths have been proven by autopsy to be due to thromboses. This makes it probable that an even higher percentage of smaller thromboses are due to polycythemia.

Ophthalmologists must realize that not only polycythemia vera, but also transient, relatives states of polycythemia may contribute to thromboses that affect the vital organs.

COURSE 6

Room 712 Periods W-4, W-5

COURSE 7

Room 712 Periods Th-4, Th-5

PAUL BOEDER, Ph.D.
Southbridge, Mass.

Aniseikonia and Space Eikonometry

A two-period course—$4.00

This two-hour course is being presented twice, as Course 6 on Wednesday and as

Course 7 on Thursday. **Please select course number according to day on which you wish to attend.**

First, a brief review of aniseikonia will be given, including a discussion of its causes, symptoms, incidence, iseikonic lenses and clinical results obtained.

The effects of aniseikonia on binocular space perception will then be explained, demonstrated and developed into the principles of space eikonometry. The space eikonometer and methods of measurement will be described and demonstrated, and an opportunity given for actual practice of the newest eikonometric methods with the latest instruments.

COURSE 8

Room 795 Periods M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5

HAROLD W. BROWN, M.D., et al
Eye Muscles

An eight-period course—$16.00

M-4 & 5—Physiology and Pathology of the Ocular Muscles

Harold W. Brown, M.D.
New York, N. Y.

The physiologic action of the individual muscles, conjugate and disjunctive movements (convergence, divergence) will be discussed and illustrated. From these normal physiologic acts, the pathologic variations are developed and classified. Various methods of diagnosis, mesaurements of deviation and the surgical and nonsurgical correction of these deviations will be presented.

T-4 & 5—Congenital Anomalies

Rudolf Aebli, M.D.
New York, N. Y.

The subject matter will cover the more usual congenital anomalies such as retraction syndrome and its variations, strabismus fixus, Marcus Gunn jaw winking syndrome, paralyses of both elevators of one eye with true and pseudoptosis, and also congenital individual muscle anomalies. Differential diagnosis and treatment, if any, will be discussed.

W-4—Tests

Harold W. Brown, M.D.

Tests in common use will be briefly described and the relative merits discussed. Emphasis will be placed on the screen cover test and its modifications. A practical routine muscle test will be outlined and the technic of the various procedures will be described in detail.

W-5—Surgical Technics in the Management of Motor Anomalies

William B. Clark, M.D.
New Orleans, La.

A thorough understanding of the anatomy and physiology of the ocular muscles is essential before an effective technic in the surgical treatment of motor anomalies can be perfected. A discussion of the dissection of tissues in layers, the preservation of Tenon's capsule covering the muscle, the severing of check and lateral ligaments, the use of suture materials for reattaching muscles to the sclera, and closure of the wounds will be discussed in detail.

Th-4 & 5 — Surgery of the Extraocular Muscles

Harold W. Brown, M.D.
and
Rudolf Aebli, M.D.

The indications and contraindications for surgical correction of heterophoria and heterotropia will be discussed. Special emphasis will be placed on the methods used in selecting the proper muscle or muscles to be operated on and the amount of correction that can be expected from the different types of surgical procedures.

COURSE 9

Room 711 Periods M-5, M-6

WILLIAM B. CLARK, M.D.
New Orleans, La.

Surgery to Ocular Muscles

A two-period course—$4.00

A review of the surgical anatomy and the physiology of the ocular muscles will be presented. Effort will be made to rationalize the use of certain procedures with the anatomy and physiology of the eye muscles. The selection of the type of procedure to plan to follow for certain anomalies will be discussed along with the preliminary preparation of the patient for surgery, the choice of the anesthetic agent, and the postoperative care of the patient.

COURSE 10

Room 793 Periods M-4, M-5, T-4, T-5

COURSE 11

Room 793 Periods W-4, W-5, Th-4, Th-5

JACK C. COPELAND
Chicago, Ill.
BY INVITATION

Streak Retinoscopy

A four-period course—$8.00

This four-hour course is being presented twice, as Course 10 on Monday and Tuesday and as Course 11 on Wednesday and Thursday. **Please select course number according to days on which you wish to attend.**

The following outline describes the steps of the demonstration.

1. Determination of the neutral point.
2. Revealing low astigmatic errors.
3. Locating the zone of the astigmatic band.
4. Determining the power of the cylinder.
5. Estimating the type and degree of error without lenses.
6. "Guide Angle" technic, in refining axis position.
7. Plus cylinder procedure.

COURSE 12

Room 710 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

FRANK D. COSTENBADER, M.D.
Washington, D. C.

HERMANN M. BURIAN, M.D.
Iowa City, Iowa

MARSHALL M. PARKS, M.D.
Washington, D. C.

Concomitant Strabismus: Physiology and Management

A twelve-period course—$24.00

This course will deal with the physiology of binocular alignment and binocular vision with emphasis on both the sensory and motor abnormalities which take place when binocularity is no longer possible. On this foundation, the diagnosis and management of concomitant strabismus will be discussed.

COURSE 13

Room 705 Periods Th-5, Th-6

A. GERARD DeVOE, M.D.
New York, N. Y.

The Clinical Application of Gonioscopy

A two-period course—$4.00

This course will review the fundamentals of gonioscopy as well as controversial points.

Anatomy and instrumentation will be described in detail. Attention will then be directed to important clinical conditions in which gonioscopy may be of value. These include injuries, all types of glaucoma, uveitis, tumors, congenital anomalies, preoperative and postoperative examination of certain eyes.

COURSE 14

Room 777 Periods M-5, M-6

HERMAN ELWYN, M.D.
New York, N. Y.

Heredodegenerative Diseases of the Retina

A two-period course—$4.00

The meanings of the terms "abiotrophy," "heredodegeneration" and "heredoconstitutional" are discussed. An attempt is made to classify the heredodegenerations of the retina on the basis of the individual elements of the retina.

COURSE 15

Room 784 Periods T-5, W-5, Th-5

WALTER H. FINK, M.D.
Minneapolis, Minn.

Management of Oblique Muscle Problems

A three-period course—$6.00

The presentation will be divided into two parts:

I. Management of superior oblique problems

II. Management of inferior oblique problems

Each part will be considered under the following headings:

a. Importance of the defect as an obstacle to binocular action

b. Diagnostic criteria
   1. The selection of efficient tests and correct interpretation has led to more accurate diagnosis

2. More accurate diagnosis has demonstrated that the defects are more frequent than formerly believed
3. Principles governing diagnosis
4. Diagnostic procedures
   x. Basis of selecting tests
   y. Description of tests proved most effective

c. Principles involved in management
1. Early treatment essential
2. Refraction
3. Prism
4. Fusion training
5. Surgical treatment
   x. Indications
   y. Dangers
   z. Management of the various types of problems encountered

d. Anatomical factors which determine the surgical technic (muscle-insertion-adjacent structures)

e. Effective surgical procedures
1. Discussion of various methods
2. Evaluation of results

f. Present status of problem

Current opinion and personal experience will be considered. Cases will be cited to illustrate points discussed.

COURSE 16

Room 732 Periods M-4, T-4, W-4, Th-4

LeGRAND H. HARDY, M.D.
New York, N. Y.

EDMOND L. COOPER, M.D.
Detroit, Mich.

DANIEL SNYDACKER, M.D.
Chicago, Ill.
and

ELIZABETH K. STARK
New York, N. Y.
BY INVITATION

Basic Principles of Orthoptics

A four-period course—$8.00

This course will consist of three lecture-discussion periods and one practical discussion seminar. The basic laws of learning, especially as they relate to the acquisition of motor skills, will be presented, the methods and results of orthoptics evaluated (Hardy); the special problems presented by children will be discussed (Cooper); the different nature of the adult problem and methods for meeting it will be presented (Snydacker); and one session will be devoted to the analysis of the most common problems and pitfalls encountered in office and clinical orthoptics with methods of solving them (Stark).

COURSE 17

Room 734 Periods W-5, W-6

DONALD W. HASTINGS, M.D.
Minneapolis, Minn.
BY INVITATION

Psychosomatic Medicine in the Practice of Ophthalmology and Otolaryngology: Application of Principles

A two-period course—$4.00

This discussion is intended to review the basic principles involved in the development of psychosomatic disorders and to discuss some methods of therapy which can be used by the physician who has no extensive training in psychiatric principles and practice. The eye, ear, and upper respiratory tract are common seats for the expression of emotional difficulties.

The position is taken that the majority of psychosomatic symptoms are determined by emotional conflicts which create anxiety. Anxiety, like physical pain, is an unpleasant sensation, and to maintain homeostasis, the body must try to be rid of it. Symptoms are interpreted in the main as defenses against the anxiety arising from emotional conflicts. Frequently the patient is not aware, or is only dimly aware, of the factors involved in the emotional conflicts within himself. Principles of therapy which can be utilized by the ophthalmologist and otolaryngologist will be discussed.

COURSE 18

Room 734 Periods M-4, M-5

COURSE 19

Room 713 Periods Th-4, Th-5

JOHN W. HENDERSON, M.D.
Ann Arbor, Mich.

Perimetry

A two-period course—$4.00

This two-hour course is being presented twice, as Course 18 on Monday and as Course 19 on Thursday. **Please select course number according today on which you wish to attend.**

The basic principles of perimetry together with their clinical applications using ac-

tual patient case reports and field examples. Localization along the visual pathway will be stressed. There will be a short discussion of the value of flicker fusion perimetry as a supplement to the usual methods.

COURSE 20

Room 752 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, Th-4, Th-5

WENDELL L. HUGHES, M.D., et al

Plastic Surgery in Ophthalmology

A ten-period course—$20.00

This course is designed to stimulate the interest of the general ophthalmologist in reparative surgery around the eye. Many of the common injuries will be illustrated by means of slides and motion pictures to show the technics of repair of conditions that will be met with in ophthalmologic practice.

M-4 & 5—A. Gerard DeVoe, M.D.
New York, N. Y.

General discussion of fundamentals of various facts that are related to the reparative surgery of the lids and adnexa. This will include pertinent anatomy, recommendations on choice of tissues for replacement, the various types of sutures, instruments, dressings and medications that are used in operative work and dressings.

M-6— Deane C. Hartman, M.D.
Los Angeles, Calif.

The discussion will include removal of pterygiums, pseudopterygiums, symblepharon, conjunctivoplasty, fornix reconstruction and ptosis.

T-4— Joseph G. Hill, M.D.
Toronto, Ont., Canada

Dr. Hill will discuss technics used in repair of entropion and trichiasis, as well as the proper handling of chalazia.

T-5 & 6— Byron Smith, M.D.
New York, N. Y.

Lid reconstruction for loss of various portions of the lids, canthoplasty and epicanthus will be discussed.

W-4 & 5—Wendell L. Hughes, M.D.
Hempstead, N. Y.

The discussion will include repair of lacerations and ectropion, conditions of the lacrimal apparatus and enucleation and evisceration.

Th-4 & 5—Alston Callahan, M.D.
Birmingham, Ala.

Dr. Callahan will discuss implants, socket reconstruction and fractures, immediate and late repair.

COURSE 21

Room 726 Periods W-5, W-6

FRITZ JARDON
Detroit, Mich.
BY INVITATION

Fitting of Ocular Prostheses

A two-period course—$4.00

An entirely new concept of eye fitting will be discussed over enucleated sockets as worked out by personal experience in fitting sockets, covering the whole field of surgical procedures.

A series of conformers will be shown by which a difficult-to-fit socket, underdeveloped eyes, and shallow fornices can be reconstructed to approximate normal function.

Present and future developments in the field will be discussed.

COURSE 22

Room 705 Periods W-5, W-6

COURSE 23

Room 702 Periods Th-4, Th-5

LORAND V. JOHNSON, M.D.
Cleveland, Ohio

Aqueous Tonography

A two-period course—$4.00

This two-hour course is being presented twice, as Course 22 on Wednesday and as Course 23 on Thursday. **Please select course number according to day on which you wish to attend.**

In the first part of the period the principles on which tonography is based will be discussed. Open and closed manometer calibration of tonometers will be defined. The deformation of the cornea and distention of the globe during tonometry, and their relationship to tables of ocular volumes, will be described. The variation in aqueous flow with intraocular pressure will be discussed and these concepts used to calculate facility of outflow and rate of aqueous flow from tonographic data.

In the second part of the period the apparatus will be demonstrated on a patient, the record obtained measured and the results calculated. The period will con-

clude with the presentation of typical results from several types of glaucoma.

COURSE 24

Room 728 Periods M-5, M-6

COURSE 25

Room 728 Periods T-5, T-6

VINCENT C. JOHNSON, M.D.

BY INVITATION

and

ROBERT H. DAVIES, M.D.

Detroit, Mich.

Diagnostic Value of X-ray in Ophthalmology

A two-period course—$4.00

This two-hour course is being presented twice, as Course 24 on Monday and as Course 25 on Tuesday. **Please select course number according to day on which you wish to attend.**

Roentgenograms properly made will demonstrate the osseous structures about the orbits in detail. Multiple projections have been designed to show structures in this area to the best advantage; the technics commonly employed will be discussed, and resulting roentgenograms will be displayed. The interpretation of such roentgenograms, of course, requires knowledge of roentgen anatomy. For this reason a part of our discussion will be devoted to a detailed explanation of the various shadows that may be observed in the roentgen studies of this region. For the sake of completeness, a brief discussion of the roentgen anatomy of the entire skull will be included.

In addition to discussion of the normal skull and orbital areas, multiple pathologic situations will be adequately illustrated and discussed. It is known that the roengenogram is capable of displaying fractures about the orbits which may result in alteration of orbital contents. Roentgen opaque foreign bodies in the eye can be identified and accurately localized by multiple roentgen methods. Every effort will be made to completely describe and display such traumatic situations.

Since it is believed that abnormalities in the paranasal sinuses adjacent to the orbital areas may affect the eye, we shall briefly demonstrate and discuss the roentgen signs of pathologic changes involving these cells.

A considerable portion of the discussion will be devoted to description and illustration of the roentgen manifestations of intracranial lesions which may be located in all parts of the head, since the ophthalmologist is frequently called upon to aid not only in the diagnosis but also in the localization of such lesions. Some intracranial lesions may be recognized by localized bone changes or by calcification within these lesions; others require additional studies, such as encephalograms and ventriculograms. Arteriography has also proved to be of assistance in this localization in certain selected instances. Numerous roentgenograms of localizing value in multiple cases will be shown on slides.

Occasionally the intraorbital structures may be altered, chiefly by calcification, which can be recognized on the roentgenograms, and some of these situations will be included and illustrated.

COURSE 26

Room 786 Periods M-5, M-6, T-5, T-6, W-5, W-6, Th-5, Th-6

WILLIS S. KNIGHTON, M.D., et al

Highlights of Glaucoma

An eight-period course—$16.00

A discussion of glaucoma designed to encompass the various concepts of etiology, classification and management. Basic principles will be outlined, leaving time for the student to participate in a roundtable discussion.

M-5—Classification and Diagnosis
Willis S. Knighton, M.D.
New York, N. Y.

M-6—Wide-angle Glaucoma
Edwin B. Dunphy, M.D.
Boston, Mass.

T-5—Narrow-angle Glaucoma
Paul A. Chandler, M.D.
Boston, Mass.

T-6—Secondary Glaucoma
A. Gerard DeVoe, M.D.
New York, N. Y.

W-5—Pathology
Joseph A. C. Wadsworth, M.D.
New York, N. Y.

W-6—Medication
P. Robb McDonald, M.D.
Philadelphia, Pa.

Th-5—Surgery
Harold G. Scheie, M.D.
Philadelphia, Pa.

Th-6—Round Table Discussion

COURSE 27

Room 702 Periods M-4, T-4, W-4

HEDWIG S. KUHN, M.D.
Hammond, Ind.

Industrial Ophthalmology

A three-period course—**$6.00**

M-4—What constitutes an over-all modern eye program? A discussion of choice of visual program according to type and size of industry; discussion of visual testing in industry, visual skills recorded, instrumentation used, policies and objectives of testing programs; discussion of visual skills as matched to job demands in various industries; color and illumination (illustrated with slides).

T-4—Eye Injuries: Types of injuries; full discussion of elements of successful eye protection programs; handling of injuries in plant dispensary by nurses and attendants; handling of injuries in ophthalmologist's office (illustrated by slides).

W-4—Eye Correction Problems and Objectives: On what basis employees are referred out for correction; what constitutes occupational correction; where, how and when are refractions carried out. A fully illustrated discussion of visual skills and their relationship to optical aids and refractive errors corrected by safety or regular glasses.

COURSE 28

Room 776 Periods W-4, W-5, W-6
Th-4, Th-5, Th-6

JAMES E. LEBENSOHN, M.D., et al
Refraction

A six-period course—$12.00

W-4 & 5—Basic Concepts
William F. Moncreiff, M.D.
Chicago, Ill.

W-6 & Th-4—Special Problems in Children
S. V. Abraham, M.D.
Los Angeles, Calif.

Th-5 & 6—Presbyopia
James E. Lebensohn, M.D.
Chicago, Ill.

COURSE 29

Room 727 Periods W-5, W-6

COL. AUSTIN LOWREY, JR. (MC)
Honolulu, Hawaii

Military Ophthalmology

A two-period course—$4.00

A discussion of the number and types of eye casualties and associated injuries. Types of missiles, contusion and concussion agents, bacteriologic warfare agents, agents producing burns (including chemical warfare agents) and atomic weapons.

A discussion of the echelons of medical care for the eye casualty, including first aid treatment, evacuation, definitive care, disposition and rehabilitation. A statistical review of the best methods in caring for large groups of cases (ocular contusions and their complications, intraocular foreign bodies, burns, enucleations and ocular implants).

COURSE 30

Room 728 Periods M-4, T-4
W-4, Th-4

DONALD J. LYLE, M.D.
Cincinnati, Ohio

Neuro-Ophthalmology

A four-period course—$8.00

The oculogyric system, its anatomy, function and dysfunction, will be discussed, including:

1. Binocular vision
2. Supranuclear eye movements
   a. Voluntary
   b. Involuntary
   c. Reflex
3. Nuclear and infranuclear eye movements
4. Pupillary action: autonomic control

Emphasis will be placed upon clinical application with discussion of symptoms and syndromes illustrated by case reports.

COURSE 31

Room 796 Periods M-4, M-5, M-6, T-4,
T-5, T-6, W-4, W-5,
W-6, Th-4, Th-5, Th-6

JOHN M. McLEAN, M.D.
New York, N. Y.

JACK S. GUYTON, M.D.
Baltimore, Md.

A. E. MAUMENEE, M.D.
San Francisco, Calif.

Cataract Surgery

A twelve-period course—$24.00

A course on the technic of intracapsular cataract extraction. It includes detailed discussion of preoperative and postoperative management, anesthesia and akinesia, various types of incisions and sutures, management of iris, methods of intracap-

sular lens delivery, and indications. Operative and postoperative complications. Motion picture and lantern slide demonstrations.

COURSE 32

Room 727 Periods T-5, T-6

RAYMOND E. MEEK, M.D.
New York, N. Y.

Surgical Anatomy of the Orbit

A two-period course—$4.00

The importance of anatomy in ophthalmic surgery is understood by everyone who ever performed an eye operation. Even the simplest incision for a chalazion calls for a knowledge of the location of the gray line, the tarsus, the position of the meibomian glands as well as the bulbs of the hair follicles of the lashes. Failure to know these tissues and their relationships may occasion untoward results. Likewise in the more delicate operations at the angle of the anterior chamber of the eye, a lack of knowledge of the anatomic relationships may be embarrassing. A thorough knowledge of the anatomy of the eye and orbit is the first requisite not only of a skillful operator but of every worker in the field of ophthalmology.

COURSE 33

Room 744 Periods M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5

SAMUEL J. MEYER, M.D., et al

Glaucoma

An eight-period course—$16.00

The fundamentals of glaucoma will be elaborated upon with considerable emphasis placed on the essentials of the early diagnosis. Both the medical and the surgical treatment will be given in minute detail including the use of the newer drugs that are being tried out both experimentally and clinically. The various newer diagnostic aids as advocated in the recent literature will be given considerable thought.

M-4—The Glaucomas: Definition, Mechanisms, Classifications
Peter C. Kronfeld, M.D.
Chicago, Ill.

M-5—The Histopathology of Acute and Chronic Glaucoma
Brittain F. Payne, M.D.
New York, N. Y.

T-4—Wide Angle or Compensated Glaucoma
Samuel J. Meyer, M.D.
Chicago, Ill.

T-5—Narrow Angle Glaucoma
Joseph S. Haas, M.D.
Chicago, Ill.

W-4—Gonioscopy
H. Isabelle McGarry, M.D.
Evanston, Ill.

W-5—Glaucoma Secondary to Uveitis
F. Bruce Fralick, M.D.
Ann Arbor, Mich.

Th-4—The Principles of Nonsurgical Treatment of Glaucoma
Irving H. Leopold, M.D.
Philadelphia, Pa.

Th-5—The Principles of Surgical Treatment of Glaucoma
Samuel J. Meyer, M.D.

COURSE 34

Room 781 Periods M-5, M-6

KENNETH N. OGLE, Ph.D.
Rochester, Minn.
BY INVITATION

Essential Aspects of Binocular Vision

A two-period course—$4.00

These lectures present a well-rounded review not only of the basic physiology and optics involved in normal binocular vision, but also of the existing problems. To be considered are the sensory and motor aspects of the organization of the two eyes, the basic facts of fusion, fusional movements, phorias, fixation disparity, prism vergences, stereopsis, and elements of binocular spatial localization.

COURSE 35

Room 777 Periods T-5, T-6

D. H. O'ROURKE, M.D.
Denver, Colo.

Manifest and Cycloplegic Refraction with Cross Cylinder and Astigmatic Dials

A two-period course—$4.00

This course is a practical demonstration of the use of the Jackson cross cylinder and the Lancaster astigmatic dials. Both of these methods lend themselves to the subjective examination of the eye in a simple and practical manner.

Each method may be utilized satisfac-

torily either with or without the use of a cycloplegic. Either method may be used nicely as a check on the other, and both methods are used at the time of the refraction interchangeably.

A minimum of theory will be used as necessary to emphasize the practical demonstration.

COURSE 36

Room 784 Periods T-4, W-4, Th-4

R. TOWNLEY PATON, M.D.
New York, N. Y.

Corneal Surgery

A three-period course—**$6.00**

Main emphasis will be placed on the selection of cases for both the penetrating and nonpenetrating types of corneal transplant operation. Material from several hundred kodachrome slides of operative cases will be used for demonstration. The technic of the penetrating and nonpenetrating types of transplant will be discussed in detail. In order to demonstrate the operative technics, serial slides made from drawings will be used. Other subjects dealt with will be the postoperative care of patients, complications and how to deal with them. Finally, special cases and indications for reoperation will be mentioned.

COURSE 37

Room 736 Periods M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5

BRITTAIN F. PAYNE, M.D., et al

Practical Eye Pathology

An eight-period course—**$16.00**

M-4—Pathology of Detachment of the Retina

Joseph A. C. Wadsworth, M.D.
New York, N. Y.

In discussing the pathology of detachment, the greater part of the time will be taken up with the usual serous type occurring as a result of a break. For simplicity the subject will be divided into three headings:

1. Detachment following trauma — that following a direct blow to the eye
2. Detachment following an intraocular tumor
3. Serous type detachment

Eyes that have been badly disorganized by perforating injuries will not be included in discussion of detachment of the retina following trauma. Such eyes are lost because of the severe damage of the inner structure of the eye and the detachment of the retina is comparatively minor damage. Eyes that have been subjected to direct trauma oftentimes develop a detachment of the retina some time later. Such cases will be discussed and an effort will be made to explain the process and pathology causing the detachment. Microscopic sections will be shown to document statements made. The importance of a detachment following trauma and a clear picture of the sequence of events that take place following such injury to the eye is of great interest from the medicolegal standpoint.

The pathology of detachment following intraocular tumor will be discussed and microscopic sections will be used to demonstrate certain important parts. The actual detachment in an eye containing an intraocular tumor is of secondary importance; however, a discussion of differential diagnosis will be made and some pathologic interpretations will be demonstrated to help explain some of the clinical findings.

M-5—Pathology of Intraocular Tumors

Joseph A. C. Wadsworth, M.D.

A brief course on the pathology of intraocular tumors will be presented from microscopic sections and an effort will be made to associate clinical findings with the microscopic picture.

Histologic and clinical signs will be shown to aid in differential diagnosis of the benign and malignant types of pigmented tumors. Individual cases will be described to document certain phases of the neoplasms and a classification of malignant melanomas will be presented, indicating the histology as well as the prognosis in the different types.

A discussion of retinoblastoma will be given. The histology will be shown in its various forms with a short discussion correlating pathologic findings with the clinical pictures and prognosis. Astrocytomas will also be described.

Less common tumors of the uvea will be presented such as hemangiomas, neurofibromas, and metastatic tumors.

Individual sections will be furnished for study during the lecture when possible.

T-4—Pathology of Injury of the Eye

Merrill J. Reeh, M.D.
Portland, Ore.

Because such a large number of eyes are enucleated as the result of trauma and subsequent complications, it is of extreme value to consider the numerous pathologic

changes which may be encountered. The symptoms, findings, treatment and clinical course will be correlated carefully with the histopathology. Following the projection of slides and a comprehensive discussion of the subject, representative slides will be passed to the class members for study with the microscope. The presentation will not be didactic but rather a correlation between histopathology and clinical ophthalmology. Informal discussion by the class members will be encouraged.

T-5—Pathology of Tumors of the Lids and Adnexa

Merrill J. Reeh, M.D.

Tumors of the lids and adnexa are commonly encountered. Their clinical management is occasionally faulty due to improper recognition of the tumor, or to employment of incorrect or inadequate treatment. Knowledge of the clinical appearance, growth, characteristics and histopathology of these tumors must be constantly accessible to the clinician. A variety of slides representing the clinical entities and histopathology will be projected. This will be accompanied by a comprehensive discussion correlating the clinical picture and the histopathology. Following this, representative slides will be passed out to the class members for study with the microscope. Class discussion will be encouraged.

W-4—Pathologic Complications from Intraocular Operations

Brittain F. Payne, M.D.
New York, N. Y.

Complications following intraocular operations are best studied in the laboratory following the enucleation of the diseased eye. Fortunately, very few eyes are submitted to the laboratory due to the marked improvement in surgical procedures and treatment in the past few years.

Examination of ocular preparations in the laboratory offers valuable evidence concerning the failure of various intraocular operations. A microscopic study often shows certain technical errors which may be corrected in future operations. In many cases, microscopic examination will determine conditions existing before the operation and account for the loss of the eye.

A review of the globes enucleated after intraocular operations at the New York Eye and Ear Infirmary revealed that loss of eyes after glaucoma surgery exceeded all other types combined. Enucleations after cataract extractions, removal of intraocular foreign bodies and retinal detachment operations followed in about that order.

W-5—Granulomas of the Eye

Brittain F. Payne, M.D.

Granulomas affect the eye in the same manner as other parts of the body. Actually, ophthalmologists are concerned mainly with tuberculosis, sympathetic ophthalmitis, sarcoidosis, syphilis and leprosy.

With the introduction of new antibiotics, sulfonamides, and hormonal therapy, reports of favorable response in each of these conditions have been issued. It is hoped that further investigations will give us the cure of these baffling diseases of the eye.

In order to treat granulomas intelligently, it is necessary to understand their architectural components.

Emphasis should be placed on the microscopic appearance of tuberculosis and sympathetic ophthalmitis of the uvea. Demonstrations of leprosy and syphilis are included with sarcoidosis.

Th-4—Glaucoma

Bernard A. Roberts, M.D.
New York, N. Y.

The clinicopathological approach will be stressed in the demonstration of glaucoma as it is associated with various conditions, i.e., hydrophthalmus and buphthalmus, glaucoma-capsulare, endophlebitis obliterans, essential iris atrophy, malignant melanoma, Sturge-Weber's disease, diabetes, retrolental fibroplasia, retinal detachment, epithelial cyst of the anterior chamber, sympathetic ophthalmia.

Not only will the pathogenesis be discussed and demonstrated but rationale of therapy based on the underlying pathology will be explained.

Th-5—Pathology of Fibroblastic Proliferation

A. Marvin Gillman, M.D.
New York, N. Y.

A number of ocular diseases are characterized in some stage of their pathogenesis by prominent fibrovascular membrane formation. In many such states it is the fibrovascular membrane and its consequential pathology that spell ultimate loss of visual function and often beginning deterioration of the eye. Though these conditions are of differing etiologies, they have in common some injurious incident, either inflammatory, hemorrhagic, exudative or traumatic, which stimulates the reticulo-endothelial system in the ocular tissues to respond with a proliferative repair process which often seems to go beyond the safety line

for normal or near normal ocular function.

Consideration will be given to the nature, origin, development and final state of tissue organization of the fibrovascular membrane formations in the specific disease states mentioned above.

COURSE 38

Room 727 Periods M-5, M-6

RALPH H. PINO, M.D.
Detroit, Mich.

Problems of Bifocal Lenses and of Presbyopia

A two-period course—$4.00

Bifocal problems confront most of the population over 45 years of age. To the ophthalmologist, the problem comprises the combined difficulties of the patient's state of mind, ocular pathology, muscle balance, refractive errors, anisometropia, and occupation. In addition, there is the difficulty arising from the failure of some manufactured lenses to reduce aberration to a minimum.

Through research substantiated by experience, information will be presented in this course which is of substantial aid to the patient, the ophthalmologist, and to the manufacturer of bifocal lenses.

COURSE 39

Room 716 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

HUNTER H. ROMAINE, M.D., et al

Ocular Motility

A twelve-period course—$24.00

It is our desire to cover the subject, starting with the fundamentals and working through to surgical technics and an evaluation of all methods of treatment.

M-4—Applied Anatomy and Physiology of the Ocular Muscles

Hunter H. Romaine, M.D.
New York, N. Y.

Anatomy and physiology of the ocular muscles will be presented, bringing out the relationship of the anatomy and physiology to the pathologic situations that result from discrepancies in the motor system.

M-5—Etiologic Factors in Muscular Imbalance

Abraham Schlossman, M.D.
New York, N. Y.

Etiologic factors relating to the major causes of muscular imbalance will be discussed.

M-6—Classification of Motor Anomalies

Louis J. Girard, M.D.
Oyster Bay, L. I., N. Y.

Motor anomalies will be classified according to several methods and the classification related to methods of handling the defects.

T-4—Evaluation of Tests in the Diagnosis of Motor Anomalies

Edward A. Dunlap, M.D.
New York, N. Y.

An evaluation will be made of the routine tests and diagnoses and their application.

T-5—Preoperative Orthoptic Routine and Therapy

Dorothy Parkhill
New York, N. Y.
BY INVITATION

Preoperative orthoptic routine will be outlined and also methods used when operative procedure is not contemplated.

T-6—Indications and Contraindications for Surgical Intervention

Charles N. Romaine, M.D.
Richmond, Va.

A discussion of the indications and contraindications for surgical intervention will be presented.

W-4—Choice of Surgical Procedure

Byron Smith, M.D.
New York, N. Y.

The choice of surgical procedures and choice of individual procedures for certain situations will be discussed.

W-5—Surgical Technic

Truman L. Boyes, M.D.
New York, N. Y.

An evaluation of the various surgical technics will be made, including discussion of sutures and surgical materials.

W-6—Surgical Complications and Secondary Surgery

Hunter H. Romaine, M.D.

The complications following surgical intervention and suggestions on secondary operations will be considered.

Th-4—Analysis of Operative Results and Evaluation of Surgical Versus Nonsurgical Therapy

Richard C. Troutman, M.D.
New York, N. Y.

An analysis of operative results and an evaluation of conservative and surgical procedures will be made.

Th-5—Postoperative Orthoptic Routine and Therapy

Dorothy Parkhill

Orthoptic problems in handling the postoperative patient will be considered.

Th-6—Round Table Discussion—Panel

The last hour will be reserved for questions and discussions by the entire panel.

COURSE 40

Room 732 Periods M-5, M-6

COURSE 41

Room 732 Periods T-5, T-6

CHARLES SCHEPENS, M.D.

Boston, Mass.

BY INVITATION

The Technic of Indirect Ophthalmoscopy: Adjunct in Diagnosis of Retinal Detachment

A two-period course—$4.00

This two-hour course is being presented twice, as Course 40 on Monday and as Course 41 on Tuesday. **Please select course number according to day on which you wish to attend.**

The optical principles of direct and indirect ophthalmoscopy and the advantages and disadvantages of both methods are briefly outlined.

A new indirect stereoscopic ophthalmoscope is described, the technic of its use is demonstrated, followed by practical exercises with the instrument. It is hoped that the new indirect ophthalmoscopes will be available for this purpose.

Points concerning ophthalmoscopy, which are of great importance in the diagnosis of retinal detachment, are discussed.

COURSE 42

Room 731 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

RICHARD G. SCOBEE, M.D., et al

Eye Muscles

A twelve-period course—$24.00

M-4—Anatomy and Mechanics

Richard G. Scobee, M.D.
St. Louis, Mo.

The anatomy and mechanics of the six oculorotary muscles will be considered. Also discussed will be the anatomy of the fascia of the orbit in terms of clinical and surgical significance.

M-5—Physiology

David M. Freeman, M.D.
St. Louis, Mo.

Antagonists, synergists, and yoke muscles. The laws of Sherrington, Hering, Listing, and Donders. Primary and secondary deviation.

M-6—Neurophysiology

David M. Freeman, M.D.

Binocular factors. Pharmacology of the oculorotary muscles. Supranuclear, nuclear, and infranuclear lesions and their localization. Muscles necessary for ocular rotations; size and strength of the muscles.

T-4—Tests for Heterophoria

George T. Stine, M.D.
Columbus, Ohio

Factors affecting heterophoria. Types of test available. Comparison of tests and an evaluation of them. Testing technic for the cover test and for the Maddox rod.

T-5—Symptoms and Treatment of Heterophoria

George T. Stine, M.D.

Symptoms associated with esophoria, exophoria, hyperphoria, and cyclophoria. The treatment, both surgical and nonsurgical, of these entities.

T-6—Etiology of Heterotropia

Richard G. Scobee, M.D.

A classification of the various types of heterotropia upon an etiologic basis. A discussion of true paresis and apparent paresis. The clinical importance of classification in treatment.

W-4—Diagnostic Methods

David M. Freeman, M.D.

A consideration of the application and measurement of the angle kappa, of various methods of measuring the deviation, of ductions and vergences.

W-5—Vergence—The Case against Prisms

Richard G. Scobee, M.D.

A consideration of the probable error in the use of prisms for measuring the deviation in the six cardinal positions of gaze and a suggested substitute for this procedure.

W-6—Other Diagnostic Methods

George T. Stine, M.D.

Abnormal retinal correspondence. Head and face tilt. Inhibitional palsy. Bielschowsky's sign. Normal overaction of the inferior oblique.

Th-4—Analysis of the Case
Richard G. Scobee, M.D.

A consideration of data from the various diagnostic procedures in terms of actual diagnosis and determination of course of therapy.

Th-5—The Nonsurgical Treatment of Heterotropia
David M. Freeman, M.D.

Such measures as occlusion, refraction, atropinization, orthoptics, drugs and sedatives. The use of the cheiroscope, the kaleidoscope, etc., in suppression amblyopia.

Th-6—The Surgical Treatment of Heterotropia
Richard G. Scobee, M.D.

Strengthening and weakening operations in the surgery of heterotropia. Prediction of surgical results. Technic of the recession and the resection. Percentage of cures.

COURSE 43

Room 715 Periods M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5

M. NOEL STOW, M.D., et al

Applied Clinical Pathology

An eight-period course—$16.00

In this course clinical disease pictures will be studied from their pathologic viewpoint. The extensive clinical and pathologic material at the disposal of the lecturers has been photographed on kodachrome slides, allowing for group study and having some advantages over the use of the microscope, since more territory can be covered. Pathogenic mechanisms will be touched upon, but the greatest stress will be laid upon morphology in its relation to the observed clinical patterns.

M-4—Glaucoma
M. Noel Stow, M.D.
Washington, D. C.

M-5—Vascular Diseases
Arnold W. Forrest, M.D.
White Plains, N. Y.
BY INVITATION

T-4—Tuberculosis and Syphilis
A. Ray Irvine, Jr., M.D.
Beverly Hills, Calif.

T-5—Sarcoidosis, Brucellosis and Sympathetic Ophthalmia
A. Ray Irvine, Jr., M.D.

W-4—Epibulbar Tumors
M. Noel Stow, M.D.

W-5—Orbital Tumors
Arnold W. Forrest, M.D.

Th-4—Melanomas and Metastatic Tumors
Torrence A. Makley, Jr., M.D.
Columbus, Ohio
BY INVITATION

Th-5—Retinoblastomas and Pseudogliomas
Torrence A. Makley, Jr., M.D.

COURSE 44

Room 751 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

GEORGIANA D. THEOBALD, M.D., et al

Histopathology

A twelve-period course—$24.00

The teaching will be by lecture, microprojection and microscopes. Before attending, a review of normal histology is recommended.

Uveitis

Leonard Christensen, M.D.
Portland, Ore.

Postcataract Complications

Michael J. Hogan, M.D.
San Francisco, Calif.

Eyes removed after cataract extraction exhibit most of the basic pathologic changes which the beginner in pathology must learn to recognize. This discussion is given not to show what happens after cataract extraction but to demonstrate the variety of pathologic changes in the eye. The following conditions will be discussed:

1. Iridocyclitis
   a. Retained lens material
   b. Incarceration of lens capsule, cortex and vitreous in the wound
   c. Phaco-anaphylaxis
   d. Low grade infection
2. Iris incarceration and prolapse with their sequelae
3. Hemorrhage and its effects
4. Infection in the anterior chamber and endophthalmitis
5. Epithelial ingrowth in the anterior chamber
6. Sympathetic ophthalmia

Sympathetic Ophthalmia and Tuberculosis
John S. McGavic, M.D.
Bryn Mawr, Pa.

Sympathetic ophthalmia and tuberculosis are both granulomatous diseases. Sympathetic ophthalmia is always bilateral and tuberculosis frequently is. Histopathologic differentiation is possible; it requires ac-

curate knowledge of cellular detail. Differentiation of the two diseases is good exercise.

Sympathetic ophthalmia represents an infiltrative type of inflammation, while tuberculosis is destructive. Tuberculosis tends to have a spotty distribution while sympathetic uveitis is widespread. The granulomatous lesions (tubercles) in the two diseases are also different on histopathologic examination.

Primary Orbital Tumors

Edith Parkhill, M.D.
Rochester, Minn.
BY INVITATION

The pathologic characteristics of primary orbital tumors originating from glandular, vascular, meningeal, and nerve sheath muscle and various types of connective tissue will be discussed, as well as the importance of differentiating between the so-called mixed tumors and cylindromas of the lacrimal glands. Sections of the more commonly occurring orbital tumors will be provided for microscopic study.

Glaucoma

Theodore E. Sanders, M.D.
St. Louis, Mo.

The essential changes produced in the tissues of the eye by increased intraocular pressure will be studied. The various lesions causing secondary glaucoma will also be described. The possible mechanisms which cause both primary and secondary glaucoma will be discussed.

Inflammation

Georgiana D. Theobald, M.D.
Oak Park, Ill.

The cytology, pathology and sequelae of ocular inflammations will be discussed.

Ocular Melanomas

Helenor Campbell Wilder
Washington, D. C.

Morphologic characteristics of ocular melanomas will be correlated with statistical studies of their behavior. Histogenesis of these tumors will be discussed. Lantern slides will be used to demonstrate the histopathology.

Retinoblastoma

Levon K. Garron, M.D.
Oakland, Calif.

Retinoblastoma is a malignant tumor of the retina which was formerly called glioma. The tumor is always congenital even though it may not become manifest for some years after birth. It has been well established that there is a hereditary tendency in retinoblastoma, probably transmitted in the genes as a dominant trait. Although the incidence of one case in 34,000 births would tend to place this view in suspect, there are many instances of hereditary and familial occurrence of retinoblastoma documented in the literature. In two-thirds of the cases the tumor is recognized before the third year of life; however, some cases have not appeared until the age of ten and later. The tumor has been said to be bilateral in 25 per cent of cases, but recent studies of this disease suggest a much higher figure. The second eye is affected spontaneously on the basis of multiple origins and not through metastasis. It is characteristic for the tumor to spread by extension, although metastasis to distant points via the blood stream have occurred.

COURSE 45

Rooms 788 & 789 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

HARVEY E. THORPE, M.D.
Pittsburgh, Pa.

ROBERT J. MASTERS, M.D.
Indianapolis, Ind.

ALEXANDER A. KRIEGER, M.D.
Pittsburgh, Pa.

JOHN MELVIN MASTERS, M.D.
Indianapolis, Ind.

CAPT. SAMUEL N. KEY, JR., USAFR (MC)
Keesler Air Force Base, Miss.

Slit Lamp Biomicroscopy

A twelve-period course—$24.00

The lectures and demonstrations of this course are planned for beginners in slit lamp microscopy and for those registrants who desire a review of the elementary features and new advances in biomicroscopy.

The early part of each session is devoted to an illustrated lecture, which is followed by a period of actual work with the slit lamps by the members of the class.

The illustrated lectures cover, in regular order from day to day, studies of the normal and pathologic cornea, anterior chamber, iris, crystalline lens, and vitreous humor. Special attention is given to the findings which can be made with no other instrument, a factor which makes the slit lamp indispensable in the routine practice of ophthalmology. The newer application of the slit lamp with gonioprism and the recent advances in technic for examination of the deep vitreous and retina will be explained.

The demonstration period is utilized to make each class member familiar with the manipulation of the instruments. During each demonstration period a round-table question-and-answer discussion is carried on for the class members who are already familiar with the instruments.

COURSE 46

Room 722 Periods M-5, M-6

DERRICK VAIL, M.D.
Chicago, Ill.

HENRY MINSKY, M.D.
New York, N. Y.

The Zonular Membrane and Cataract Surgery

A two-period course—$4.00

During the first hour a motion picture on "Anatomy of the Zonular Membrane and the Posterior Canal" will be shown and described by Dr. Minsky. This will be followed by Dr. Vail's lecture on "Operation of Choice in Cataract Surgery," in which the mechanics of the operation according to anatomic arrangement of the zonular membrane will be pointed out.

COURSE 47

Room 702 Periods M-5, M-6, T-5, T-6, W-5, W-6

MAYNARD C. WHEELER, M.D.
and
PHILIP KNAPP, M.D.
BY INVITATION
New York, N. Y.

Diagnosis and Treatment of Concomitant Strabismus

A six-period course—$12.00

The practical handling of concomitant strabismus will be fully discussed. This will include methods of examination, measurement of the deviation, diagnosis and sensorial evaluation of convergent and divergent strabismus. The medical treatment, the indications for surgery, the choice of operation and the postoperative treatment will be considered in detail.

COURSE 48

Room 784 Periods M-6, T-6, W-6

MEYER WIENER, M.D.
Coronado, Calif.

Practical Points in Surgery

A three-period course—$6.00

The course is planned to emphasize some of the vital points of technic of the various surgical procedures which will make the operations easier to perform. Application of the knowledge of anatomic structure of the tissues, their relation to other parts, as well as the physics involved, will be discussed.

COURSE 49

Room 706 Periods W-4, W-5

JONAS S. FRIEDENWALD, M.D.
Baltimore, Md.

Clinical Measurements on the Rate of Aqueous Flow

A two-period course—$4.00

An analysis of the sources of error in tonographic and fluorescine methods of measuring the rate of aqueous flow. The discussion will be chiefly mathematical and physiological.

INDIVIDUAL COURSES — OPHTHALMOLOGY

COURSE 201

Room 724 Period M-6 or T-6

MOACYR E. ALVARO, M.D.
Sao Paulo, Brazil

Uveitis: Etiology and Treatment

The various types of uveitis will be discussed, but chronic endogenous uveitis, of the granulomatous and nongranulomatous type, will be dealt with in greater detail.

The clinical diagnosis of uveitis is always easy, but it is not always possible to find out from the ocular symptoms whether the uveitis is of the granulomatous type or not, in spite of the fact that there are definite clinical symptoms for each of these types. The subjective and objective symptoms of both types of uveitis will be discussed.

The etiologic diagnosis is difficult to establish. The various examinations which should be made in order to establish the etiologic cause in granulomatous uveitis will be discussed: syphilis, tuberculosis, brucellosis, sarcoidosis, toxoplasmosis, histoplasmosis, virus diseases, etc. The tests to establish the etiology in cases of nongranulomatous uveitis will also be dealt with: hemogram, blood sedimentation, clinical examination for foci such as

teeth, tonsils, nasal sinuses, intestinal tract, gall bladder and appendix, and genito-urinary apparatus. The different sensitization tests for the several types of streptococcus, gonococcus and other rarer allergens will also be discussed. The order in which these tests should be made and the possibility of immediate treatment will also be studied.

Present day knowledge of the pathogeny of the disease will be mentioned as a basis for the discussion of the various modern methods for the treatment of uveitis and its complications.

COURSE 202

Room 781 Period T-4 or W-4

BENNETT Y. ALVIS, M.D.
St. Louis, Mo.

End Results of Various Types of Cataract Wound Closure

Proper closure of the cataract wound demands exact approximation of the wound margins and security against reopening. The factors involved are the type of incision, sutures, conjunctival flap, and iridectomy or iridotomy. The discussion will touch on these points:

Position, extent, bevel, manner of making

Sutures

Conjunctival, scleroconjunctival, corneoscleral over the surface, through lips of wound, preplaced, postplaced

Devices for sealing the wound

Conjunctival flap: limbal based; fornix based

Advantages and disadvantages of each

Fibrin coagulum

Iridectomy or iridotomy: radical or peripheral; purpose, number and position

Effects of these various features of wound closure on clinical results will be discussed.

COURSE 203

Room 750 Period M-6 or T-6

MARIO AMENABAR, M.D.
Santiago, Chile
BY INVITATION

Localization and Closure of Holes in Cases of Detachment of the Retina

To localize the tear, a specially devised Transilluminator is applied during the operation in the correspondent sector of sclera; and when the hole gets transilluminated, another instrument fixes the luminous end of the localizer, leaving a clear mark on the sclera that corresponds exactly to the situation of the tear.

Diathermy is used only when the operator knows exactly where the tear is located and can thus be circumscribed to the hole and its surrounding retinal area, leaving the eye with a minimum of coagulation.

To close the tear the scleral area of the hole is treated with a small ball electrode, making independent applications that are not strong enough to close the tear and that are reinforced by very fine penetrating applications until the hole has been surrounded by them. These intrascleral applications will not perforate the retina and aid in evacuating the subretinal fluid, checking the situation of the tear, and efficiently closing the retinal hole.

Two new instruments will be exhibited: the retinal hole localizer and the fixator-marketor.

Room 14 Period M-6

Lecture and Free Demonstration by
HAROLD F. FALLS, M.D.
Ann Arbor, Mich.
of
EMBRYOLOGY OF THE EYE
Motion Picture with Sound

Monday, October 13
4:30 p.m., Room 14

COURSE 204

Room 705 Period M-4 or T-4

WALTER S. ATKINSON, M.D.
Watertown, N.Y.

Measures Which Make Cataract Extraction Safer and Easier

For years, the goal in cataract surgery has been the complete removal of the cataract with as little disturbance to the function and appearance of the eye as possible, or, briefly, an intracapsular extraction with round pupil. Toward the attainment of this goal, simple practice methods for the introduction of sutures and the improvement of intracapsular technic will be suggested, the selection and care of the instruments will be emphasized, and recent improvements in preanesthetic medication and local anesthetic procedures will be discussed.

COURSE 205

Room 777 Period M-4 or T-4

J. MASON BAIRD, M.D.
Atlanta, Ga.

Acute Lesions of the Cornea

A discussion of the clinical aspects of acute corneal lesions including the more common type of corneal ulcers and the acute lesions that are most often seen. The basic causes and treatment with topical applications, antibiotics, chemotherapeutics, surgery and roentgen therapy will be discussed. Color slides will be shown of the lesions when first seen and after treatment. The course, duration and results will be discussed in detail.

COURSE 206

Room 722 Period M-4 or T-4

GEORGE S. BAKER, M.D.
Rochester, Minn.
BY INVITATION

Neuralgias of the Head: A Clinical Review

The term "neuralgia" has been in clinical usage for centuries. By definition neuralgia is a sharp, intermittent pain in the distribution of a given nerve without apparent cause. Our conception may change a bit during modern times when a careful neurologic or anatomic study of the case leads one to suspect that the neuralgia may be produced by some organic lesion; namely, inflammatory, metabolic, vascular, toxic or structural defect. It is true that such causes as aneurysms, localized infections and tumors have been repeatedly described as the etiologic factor in one or the other forms of major tic douloureux. Sometimes by way of an introspective psychiatric evaluation ample reasons for the so-called idiopathic pain seem well established. Whenever the cause is clear to the physician, the treatment is obviously more successful for the patient.

The differential diagnosis and the neurosurgical treatment of the major and minor neuralgias of the head will be demonstrated from a clinical point of view. Specially prepared lantern slides and colored motion pictures will be used to emphasize the close relationship of these conditions to diseases of the eye, ear, nose and throat.

COURSE 207

Room 17 Period M-5

OTTO BARKAN, M.D.
San Francisco, Calif.

Congenital Glaucoma: Diagnosis and Treatment

Corneal cloudiness, injection of the bulbar conjunctiva, increased lacrimation or blepharospasm should lead to the suspicion of glaucoma even though no enlargement of the eye has occurred. In view of the urgency of early operation, the tension should be tested with a tonometer with the patient well relaxed under ether anesthesia (mid second plane) at the moment the tension is taken. Once the diagnosis is established, surgery should be performed immediately, or if delayed, 5 per cent prostigmine should be instilled every three hours. The surgical treatment of congenital glaucoma will be reviewed and the present simplified technic of gonitomy described. A motion picture of goniotomy will be shown.

COURSE 208

Room 711 Period W-5 or Th-5

JOHN G. BELLOWS, M.D.
Chicago, Ill.

Changes in the Crystalline Lens

From the time of birth until death, the crystalline lens undergoes continuous alterations. The physical, chemical, and histologic changes that take place in the crystalline lens are followed from infancy to senility. The biomicroscopic appearances from birth to an advanced age are then described. Finally, the senile lens opacities are depicted from the incipient forms up to the fully developed stages of senile cataracts.

COURSE 209

Room 784 Period M-5
or or
Room 776 Period T-5

CONRAD BERENS, M.D.
New York, N. Y.

Etiology and Treatment of Uveitis

The etiology of uveitis will be discussed from the experimental, clinical and pathologic points of view. Especial consideration will be given to the role that sinusitis may play in the etiology.

The diagnosis of other etiologic factors such as syphilis, tuberculosis, toxoplasmosis and other diseases will be briefly outlined.

The treatment of gonorrheal, syphilitic, tuberculous, focal infection, brucellosis and toxoplasmosis uveitis will be outlined. Local treatment by heat, drugs and nonspecific therapy will be considered.

Surgical treatment of uveitis and its complication will be discussed with special reference to retrobulbar injections of alcohol and cycloelectrolysis for the control of tension.

COURSE 210

Room 722 Period T-5 or W-5

RAYNOLD N. BERKE, M.D.
Hackensack, N. J.

Surgery of the Oblique Muscles

This subject is important in the surgical correction of the vertical extraocular muscle anomalies because the oblique muscles are primarily or secondarily involved in every case of hypertropia. Therefore, every ophthalmic surgeon should be familiar with the indications and the surgical technic for these operations.

Slides and motion pictures will be shown illustrating these points.

COURSE 211

Room 727 Period W-4 or Th-4

JEROME W. BETTMAN, M.D.
San Francisco, Calif.

Value of Goniotomies

Goniotomy is definitely the operation of choice in congenital glaucoma. Eighty-two per cent are cured. Other glaucoma operations are unquestionably inferior in the treatment of this condition.

The early signs and symptoms of congenital glaucoma will be considered. The pathogenesis of congenital glaucoma and the theories of mechanism of goniotomy will be discussed.

There are five modern types of goniotomy procedures. The technic of each will be considered and the merits of each compared. Preoperative and postoperative care will be outlined.

COURSE 212

Room 727 Period M-4

COL. VICTOR A. BYRNES (MC)
Randolph Field, Texas

Adjuncts in Teaching Ophthalmology

The most successful instructional period in ophthalmology is the one which leaves the greatest amount of useful knowledge in possession of the student upon its completion. Many factors enter into the amount of information which can be imparted. The design of the classroom, the facilities provided in the classroom, the types of instructional aids used, including printed material, partially completed outlines, photographic material on slides or motion picture films, charts, drawings, tridimensional models, and blackboard drawings are all important. Combinations of these instructional aids are more effective than the use of a single type. There are errors in the use of these aids which will nullify their otherwise valuable effect. These points will be individually discussed and examples of each type will be demonstrated.

Other items to be discussed are: types of blackboards with suggestions for their use, the use of slides, type and size of lettering, proportions of graphs, use of color, relationship of data to practical applications and forms in which data may be presented.

COURSE 213

Room 711 Period W-4 or Th-4

F. PHINIZY CALHOUN, JR., M.D.
Atlanta, Ga.

Dystrophies and Degenerations of the Cornea

The more common conditions under this heading will be grouped according to the underlying pathologic process and the site of origin in the cornea in the light of our present knowledge. This approach helps to avoid prevalent confusion in terminology and simplifies the clinical recognition of each condition by more closely correlating the clinical findings with the underlying pathologic process.

The course will be illustrated with lantern slides and will include a discussion of present day methods of therapy in these conditions.

COURSE 214

Room 706 Period W-6 or Th-6

ALSTON CALLAHAN, M.D.
Birmingham, Ala.

Repair of Lid Lacerations

Varieties of lid lacerations are innumerable, but all may be simply classified for the purpose of selection of the proper reparative technic according to the direction of lid tension which has been released by the injury or which will be augmented by the reunion of the wound edges. The de-

tailed knowledge of the surgical anatomy and physiology of the eyelids must be possessed by the surgeon, and familiarity with the fundamental principles of lid repair makes the surgical reconstruction progress in a logical and systematic manner. Correct immediate repair of an injury avoids, or at least minimizes, a secondary plastic operation; and if lid injuries have previously received improper or inadequate primary care, the resulting deformity usually must be reduced to the original trauma to effect the proper restoration.

Color transparencies of a representative series of lid lacerations, with related artist's drawings and other visual aids, demonstrate carefully the performance of such technics as (1) Wheeler's halving technic, the separation of the lid into two laminas with separate management of each; (2) intermarginal lid adhesions; (3) tongue-in-groove repair; (4) methods of anastomosis of severed canaliculi.

COURSE 215

Room 750 Period W-5 or Th-5

J. V. CASSADY, M.D.
South Bend, Ind.

Dacryocystitis of Infancy

Dacryocystitis of infancy is usually the result of a failure of the lacrimal duct to become completely patent. This developmental anomaly occurs in a high percentage of infants. The developmental anatomy of the nasolacrimal duct is reviewed in photomicrographs of embryo, fetus and stillborn infants. As a result of failure of the duct to canalize if conservative management does not suffice, early irrigation and establishing patency of the lacrimal duct is advocated. The simplicity of irrigation and probing the duct, as an office procedure, is illustrated.

COURSE 216

Room 712 Period M-6 or T-6

ROBERT R. CHACE, M.D.
New York, N. Y.

Ocular Malingering

This course presents a survey of various methods for detecting ocular malingering. Tests are offered in a graded series from simple to complex. Many tests can be done with ordinary refraction equipment. More elaborate tests involve special equipment. Lantern slides and demonstrations are offered of the different tests and special equipment is shown. The problem of ocular malingering is also considered from the point of view of physical, psychological and psychiatric disease.

COURSE 217

Room 733 Period T-4 or W-4

PAUL A. CHANDLER, M.D.
Boston, Mass.

Secondary Glaucoma in Aphakia

Glaucoma in aphakia may be primary glaucoma, but is usually secondary, due to pupillary block, epithelization of the anterior chamber, or peripheral anterior synechias. Peripheral anterior synechias are brought about chiefly by the complications of delayed reformation of the anterior chamber, incarceration of iris or lens capsule in the wound, prolapse of iris and vitreous loss. Emphasis will be placed on measures designed to prevent these complications which lead to glaucoma, and the treatment of glaucoma will be discussed from a medical and surgical standpoint.

COURSE 218

Room 711 Period W-6

LOUIS S. CHOLDEN, M.D.
Topeka, Kan.
BY INVITATION

Psychiatric Aspects of Informing the Patient of Blindness

This lecture will consist of a discussion of the (1) psychological meanings of blindness to the patient, (2) psychological meanings of blindness to the ophthalmologist, (3) various methods of informing the patient of his blindness and their effects on the adjustment and rehabilitation process, (4) some aspects of predicting and evaluating the patient's reaction to the news of blindness.

Some of the secondary areas which are involved in informing the patient of blindness will be discussed. These will include problems of suicide and depression, as well as some ways of using family, friends and agencies for the blind in working with the newly blinded patient.

COURSE 219

Room 14 Period M-4

FREDERICK C. CORDES, M.D.
San Francisco, Calif.

The Surgery of Congenital Cataracts

COURSE 220

Room 750 Period M-5 or T-5

PAUL L. CUSICK, M.D.
Detroit, Mich.

Ptosis: Differential Diagnosis and Treatment

A brief review of etiology along with a classification of blepharoptosis will be given. Various types and preferred surgical treatment will be illustrated with lantern slides. A motion picture will be shown which illustrates the difficulties encountered in Blaskovicz' operation. Means of avoiding these difficulties and simplifying the operation will be illustrated.

COURSE 221

Room 722 Period W-4 or Th-4

NORMAN L. CUTLER, M.D.
Wilmington, Del.

Selection and Variety of Procedures Used in Enucleation

There are now three general types of procedures used in enucleation: buried and not mobile, mobile buried, and semi-buried or integrated.

The proper selection of the procedure to be used in enucleation depends on the experiences of the operator, the type of patient and his availability for postoperative follow-up. It is the surgeon's obligation to be familiar with the different types of implants, their advantages and disadvantages, and to give the patient the best cosmetic result possible.

COURSE 222

Room 726 Period M-6 or T-6

WINDSOR S. DAVIES, M.D.
Detroit, Mich.

Clinical Pathology in Optic Atrophy

The subject of optic atrophy will be presented from the standpoint of etiology, clinical manifestations and pathology. A classification will be presented. Differential diagnosis will be stressed and a correlation made between the clinical and pathologic findings. Treatment will be discussed.

COURSE 223

Room 713 Period M-6 or T-6

EDWARD A. DUNLAP, M.D.
New York, N. Y.

Divergence Anomalies

These anomalies consist of divergence excess and divergence insufficiency. Both are characterized by certain specific findings that serve clearly to differentiate them from anomalies of convergence. Their location in the general classification of imbalances and statistics on their relative occurrence will be given.

Symptoms of each type will be listed, and the method of diagnosis will be discussed. Typical case measurements and pictures of each type will be shown.

Treatment, both conservative and surgical, will be outlined, and tabulated results of studies on over one hundred surgically treated cases of divergence excess will be presented.

COURSE 224

Room 776 Period M-5
or or
Room 733 Period T-5

JOHN H. DUNNINGTON, M.D.
New York, N. Y.

Ocular Wound Healing

The mechanism of wound healing under normal and abnormal conditions will be reviewed after which the complications arising from minor imperfections in closure will be discussed. The reactions produced by corneoscleral sutures and those observed in wounds treated with thrombin will be presented.

COURSE 225

Room 732 Period W-6 or Th-6

JOHN A. EGAN, M.D.
Denver, Colo.

Cross Cylinder Application and Theory

This course will include a description of the use of the cross cylinder as taught by Edward Jackson. Improved models will be shown and additional uses, since Jackson's time, will be described.

Lantern slides will be shown exhibiting its practical application. Large 2-foot models of cross cylinder, trial cylinder and trial frame will be used to demonstrate general technic in refraction. Twelve different uses of the instrument, including the employment of the newer correcting cross cylinder, will be described.

A brief discussion of theories involved, controversial aspects and merit in astigmatism as opposed to other methods will be presented.

COURSE 226

Room 781 Period M-4

HAROLD F. FALLS, M.D.
Ann Arbor, Mich.

Ocular Geriatrics

A comprehensive survey of the ocular changes and disease states associated with senescence. When possible, a differentiation will be made between universally occurring disease states in the aged eye and those disease processes appearing in senescence but having no direct relationship to the passage of time.

Treatment, care, and management of the senile eye and individual will receive consideration.

COURSE 227

Room 781 Period T-5

HAROLD F. FALLS, M.D.
Ann Arbor, Mich.

Practical Application of Heredity

The purpose of this course is to present the principles of heredity so that they may be employed by the clinical ophthalmologist in his daily practice. When possible, diseases having hereditary background will be illustrated and discussed. A demonstration of pedigree taking and interpretation will be presented.

COURSE 228

Room 17 Period T-4

F. BRUCE FRALICK, M.D.
Ann Arbor, Mich.

External Ocular Manifestations of General Disease

COURSE 229

Room 726 Period M-5 or T-5

BERNARD FREAD, M.D.
New York, N. Y.

Ideal Refraction Equipment: Survey of Instruments Used in Refraction

Refraction is most important in the daily routine of every ophthalmologist. A good part of each working day is spent with details of refraction and not enough emphasis has been placed upon the tools necessary to make refraction easier, more accurate, and less time consuming.

Every major refraction instrument manufactured in the United States and in the larger optical plants of England, France, Germany, Italy, Japan and Switzerland will be discussed. Well over one hundred lantern slides will graphically display this equipment.

Attention will be given not only to elementary equipment used by the beginner but also to elaborate devices used for the objective and subjective measurement of refraction of particular interest to the experienced ophthalmologist.

The lecture will include the names and functions of various instruments and their manufacturers as well as the current cost.

COURSE 231

Room 706 Period M-6 or T-6

WILFRED E. FRY, M.D.
Philadelphia, Pa.

Differential Diagnosis of Inflammatory and Degenerative Lesions at the Posterior Pole of the Eye

The differential diagnosis of inflammatory and degenerative lesions not only must be made between these two conditions but they must also be distinguished from malignancy. In malignant conditions the diagnosis is on a different basis than when the malignancy is in the midzone or in the anterior segment of the eye. When in the midzone or anterior segment, distinction must be made between a serous and a solid detachment. When the lesions are in the posterior segment the diagnosis must be made upon the ophthalmoscopic appearance plus all other information that can be afforded by a complete examination of the patient.

The situation is different when the macula alone is involved, and when the region of the disc in involved.

COURSE 232

Room 732 Period W-5 or Th-5

WATSON W. GAILEY, M.D.
Bloomington, Ill.

Management of Complications of Cataract Surgery

An attempt will be made by the speaker to outline and discuss the principal complications which may be present preoperatively and during the cataract extraction, as well as postoperatively, and the management of such conditions in an effort to make them of no serious consequence to the surgeon in his effort to obtain good useful vision.

COURSE 233

Room 776 Period M-6 or T-6

J. CONRAD GEMEROY, M.D.
Detroit, Mich.

External Diseases of the Eye

This lecture on external eye diseases is illustrated with kodachrome slides to show the usual, as well as some unusual, diseases of the lids, conjunctiva, and cornea with discussion of their diagnosis and treatment. These illustrations will be projected stereoscopically in color on the screen.

COURSE 234

Room 740 Period W-5 or Th-5

HAROLD GIFFORD, M.D.
Omaha, Neb.

Dacryocystorhinostomy

This course will present a review of the common surgical technic used in tear sac surgery. The surgical anatomy of the region will be covered. The technic of transplantation of the tear sac will be described in detail with lantern slides and motion pictures.

COURSE 235

Room 776 Period M-4 or T-4

JOHN F. GIPNER, M.D.
Rochester, N. Y.

Recent Advances in Ophthalmology

Recent advances in ophthalmic surgery including congenital cataract surgery, round pupil cataract extraction, glaucoma surgery, and implants following enucleation or evisceration will be discussed. Mention will be made of recent advances in therapeutics.

COURSE 236

Room 724 Period W-6 or Th-6

ISADORE GIVNER, M.D.
New York, N. Y.

Associated Skin and Eye Manifestations of Systemic Disease

The appearance of lesions in both the skin and eye as manifestations of general disease makes it important to be familiar with the expressions in both systems for the better recognition and handling of systemic disorders. Among the diseases with representations in both systems, the following will be elaborated upon:

I. Phakomatoses — Bourneville's disease, Recklinghausen's, Sturge-Weber.
II. Collagen diseases—acute disseminated lupus erythematosus, dermatomyositis, scleroderma and peri-arteritis nodosa.
III. Amino acid and vitamin deficiencies.
IV. Granulomatous diseases — brucellosis, sarcoid, coccidiomycosis.
V. Viral diseases—Kaposi's varicelliform dermatitis, measles, herpes zoster, vaccinia, variola, lymphogranuloma venereum.
VI. Miscellaneous — ocular-mucous membrane syndrome of erythema multiforme, Vogt-Koyanagi, atopic dermatitis, pseudoxanthoma elasticum, and xeroderma pigmentosum.

COURSE 237

Room 17 Period W-5

EVERETT L. GOAR, M.D.
Houston, Texas

Dystrophies of the Cornea

A dystrophy is a type of degeneration due to faulty nutrition of a local or general nature. Familial dystrophies might be classed as abiotrophies of hereditary origin. The various types of dystrophies will be described as well as the best slit lamp technic for seeing them. If time remains, the technics for examining the vitreous with the biomicroscope will be explained.

COURSE 238

Room 726 Period W-4 or Th-4

ORVILLE E. GORDON, M.D.
Chicago, Ill.

Applied Anatomy of the Orbital Fascia

The orbital fascia is one integrated structure whose component parts are intimately connected and whose functions are interrelated. The fascia is also physically connected to the eye and the extraocular muscles. In any extraocular surgical procedure, the fascia is a potent factor.

The fascia will be demonstrated by means of drawings and slides. The important surgical considerations of each part will be indicated.

COURSE 239

Room 744 Period M-6 or T-6

RAYMOND J. GRAY, M.D.
Pittsburgh, Pa.

Intraocular Foreign Bodies

The subject of intraocular foreign bodies is dealt with mainly from the standpoint

of diagnosis, localization and extraction. Special emphasis is placed upon the management of the nonmagnetic type.

COURSE 240

Room 786 Period T-4

GEORGE P. GUIBOR, M.D.
Chicago, Ill.

Physiology of Ocular Muscles: Practical Application

How the neuromotor system works and how it becomes deranged are important. Ultimately we must accept the idea that abnormal ocular deviations result from deranged reflexes or from lesions affecting these pathways. The diagnosis of esotropia or exotropia, therefore, becomes symptomatic in character. Some of these deviations may disappear spontaneously, but there remain in certain cases conjugate deviations, nystagmus, spastic esotropia, paralytic esotropia, exotropia, or hypertropia, and dissociated vertical deviations.

In addition, the reflexes of fixation, fusion, reciprocal innervation and proprioception are deranged, modified, and reconditioned by such processes as summation, irradiation, and suppression. Examples of such derangements will be presented.

COURSE 241

Room 786 Period W-4

GEORGE P. GUIBOR, M.D.
Chicago, Ill.

Neuromotor Defects of Ocular Muscles from a Physiologic and Neurologic Viewpoint: Treatment

If the patient possessing a motor defect is immature, and if the disturbance is variable and not of mechanical origin, nonsurgical treatment may decrease the heterotropia in many cases. Thus, the use of atropine sulfate as a cycloplegic, prostigmine, prisms, fusion training, and occlusion for prolonged periods are desirable in realigning the visual axes and reconditioning the ocular reflexes. When mechanical defects are found, surgery is indicated.

COURSE 242

Room 786 Period Th-4

GEORGE P. GUIBOR, M.D.
Chicago, Ill.

Neurology of Ocular Muscles: Practical Application

Basic central anatomic structures for carrying impulses to the oculomotor system are the frontal and occipital cortices and the corticobulbar and corticomesencephalic paths. The peripheral portion of this system includes the nuclei and the third, fourth and sixth cranial nerves. The medial longitudinal bundle is the central correlating pathway for motor reflexes. The optic nerve, superior colliculus, and tectobulbar path subserve visual reflexes. These facts will be demonstrated by slides and drawings.

COURSE 243

Room 705 Period M-6 or T-6

S. ALBERT HANSER, M.D.
St. Louis, Mo.

Studies in Ocular Allergy

This course is designed as a practical approach to the problems of diagnosis and treatment of allergy as related to ophthalmology. Particular emphasis will be placed upon history taking and determination of etiologic factors. The technic and interpretation of conjunctival smears will be demonstrated. There will be a discussion of allergic manifestation of vernal and phlyctenular keratoconjunctivitis, followed by a diagnostic anterior chamber puncture and vitreous puncture in relationship to allergy of the inner eye.

COURSE 244

Room 777 Period W-4 or Th-4

DAVID O. HARRINGTON, M.D.
San Francisco, Calif.

Psychosomatic Phases in Ophthalmology

There are fundamental psychologic and physiologic differences between conversion symptoms, vegetative neuroses, and psychogenic organic disease. We should restrict hysterical conversion phenomena to symptoms of the voluntary neuromuscular and sensory perceptive system, and differentiate them from psychogenic symptoms which occur in vegetative organ systems, the functions of which are under the control of the autonomic nervous system.

A conversion symptom is a symbolic expression of a well-defined emotional content, an attempt at relief; although, as Freud originally expressed it, these substitutive innervations never bring full relief. The symptoms express at the same time both the repressed emotion and its rejection. Because they do not fully relieve the tension, we have a pathologic condition.

A vegetative neurosis, such as emotional

hypertension, is not an attempt to express emotion but is the physiologic accompaniment of constant or periodically recurring emotional states. The chronicity of an emotional tension alone is what makes such a condition morbid.

In considering ocular disturbances of psychogenic origin it would seem rational to divide them as follows:

A. *Ocular Conversion Symptoms*
1. Blepharospasm
2. Convergence spasm
3. Asthenopia
4. Photophobia
5. Hysterical amblyopia and amaurosis

B. *Ocular Vegetative Neuroses*
1. Ciliary spasm
2. Amaurosis fugax
3. Central angiospastic retinopathy
4. Migraine
5. Glaucoma

COURSE 245

Room 784 Period M-4

MICHAEL J. HOGAN, M.D.
San Francisco, Calif.

Pathologic Significance of Lesions of the Choroid

Fundus photographs and microphotographs will be shown to correlate the clinical and pathologic changes in diseases of the choroid. A discussion of choroiditis of various types will be presented. Choroidal changes in arteriosclerosis and hypertension will be described.

Senile macular degeneration, disciform degeneration and angioid streaks, and choroideremia and gyrate atrophy will be discussed.

The differential diagnoses and pathologic findings in benign and malignant tumors of the choroid will be explained.

COURSE 246

Room 722 Period T-6 or W-6

CHARLES E. ILIFF, M.D.
Baltimore, Md.

Beta Irradiation Therapy

Beta irradiation is ideally suited for the treatment of certain lesions of the anterior ocular segment. Three types of beta applicators are now being used. The success of this valuable therapy depends on the clinician's thorough understanding of the basic principles of irradiation, and a complete knowledge of the potentialities and dangers of the applicator used. The applicators will be discussed and dosage advised.

Dramatic results have been obtained from beta-ray therapy in both bulbar and palpebral vernal conjunctivitis. Here irradiation is used as a supplement to, and not a replacement for, allergic therapy.

Small benign tumors of the lids and anterior ocular segment can be destroyed with rapidity and ease, and the cosmetic result is excellent. In treating malignant lesions, beta irradiation may be used alone or as an adjunct to surgery.

New corneal vascularization can be controlled by beta therapy and existing vessels occluded. This may produce an improvement of vision, at times rendering a contemplated transplant unnecessary. Occlusion of the vessels increases the chances for a successful transplant.

Corneal scars in which vascularization is a prominent feature become less dense after beta therapy.

The growth of small pterygiums can be inhibited by irradiating the base and apex. After surgical removal of larger pterygiums, recurrences can be prevented.

The course of tuberculous sclerokeratitis seems to be shortened and the severity reduced, resulting in less corneal damage.

COURSE 247

Room 740 Period W-4 or Th-4

FRED W. JOBE
Rochester, N. Y.
BY INVITATION

Practical Physiologic Optics

The "getting used to" period that a patient experiences with spectacles is a period in which the patient must alter his reactions to the new environment presented by his glasses. Lenses can only distort, magnify, or displace objects in the visual field. This is done in any prescription to correct the patient's visual errors. The principles involved are fairly simple and an understanding of them will eliminate many of the troublesome difficulties which patients experience. These principles will be explained and patients' difficulties with their spectacles discussed.

COURSE 248

Room 728 Period W-6 or Th-6

DEWEY KATZ, M.D.
Hartford, Conn.

Corneal Dystrophies

The subject will be discussed in the light of recent contributions. Emphasis will be directed toward factors of clinical impor-

tance and to the dystrophies which have been given special study by the author and which have been the subjects of reports.

COURSE 249

Room 733 Period W-5 or Th-5

BERTHA A. KLIEN, M.D.
Chicago, Ill.

Clinical Pathology of Retinal Lesions

The clinical manifestations of a diseased retina and pigment epithelium and their pathogenesis in general will be discussed prior to a demonstration of the specific findings in a selected group of diseases of the retina, including diabetes, macular lesions of vascular origin and their differential diagnosis, and several other retinal diseases due to vascular pathologic processes.

COURSE 250

Room 743 Period W-6 or Th-6

WILLIAM E. KREWSON, III, M.D.
Philadelphia, Pa.

Clinical Significance of Oblique Muscles

The following topics will be discussed: anatomy, physiologic and mechanical action of the oblique muscles; their pathologic action; the effects of their derangement on the other muscles; diagnosis of oblique muscle defects; indications for surgery and choice of operative procedures.

COURSE 251

Room 17 Period W-6

PETER C. KRONFELD, M.D.
Chicago, Ill.

End Results in Glaucoma Surgery

A follow-up of glaucoma patients for a minimum period of eight years after surgery reveals four main facts: (1) gross differences in the response of the two principal types, the acute, narrow-angle and the chronic, open-angle glaucoma; (2) a strong correlation between normalization of ocular tension and stationary course of the disease in the large majority of the cases of glaucoma, (3) gross differences in the end result (in the chronic open-angle type) depending upon the stage of the disease present when the normalization of tension became effective, and (4) a greater incidence of cataract than in unoperated glaucomatous or in undiseased eyes of the same age group.

COURSE 252

Room 706 Period M-5 or T-5

PLACIDUS J. LEINFELDER, M.D.
Iowa City, Iowa

Papilledema and Optic Neuritis: Differential Diagnosis

The identification of the cause for edema of the nerve head is dependent upon factors other than the appearances of the disc. This is due to the fundamental similarity of cause of the disc changes. To understand the differential diagnosis of papilledema, it is necessary to know the physiopathology of the condition. The relationship of venous pressure, increased intracranial pressure, hyperemia, and inflammation to the occurrence of edema will be discussed.

COURSE 253

Room 734 Period M-6 or T-6

ALBERT N. LEMOINE, JR., M.D.
Kansas City, Mo.

Vitreous Opacities

The anatomy and embryology of the vitreous will be briefly reviewed. The physical and chemical properties found in the normal and pathologic vitreous will be summarized.

Specific types of vitreous opacities to be considered are: congenital remnants of the embryonic vascular system; protein coagula; synchysis scintillans; asteroid hyalitis; inflammatory exudates; blood; and retinitis proliferans.

Therapeutic measures will be discussed.

COURSE 254

Room 795 Period M-6 or W-6

CECIL W. LEPARD, M.D.
Detroit, Mich.

Eye Diseases Common to Children

Those ocular diseases occurring primarily in children constitute a group of special interest to the ophthalmologist, particularly when the method of examination or the differential diagnosis differs from that for those diseases in the adult. The technic and results of examination of premature infants will be discussed. The following syndromes, together with lantern slide demonstration of findings in the eye and orbit and pathologic photomicrographs, will be demonstrated: neuroblastoma, chloroma, Schuller-Christian's disease, and eosinophilic granuloma.

COURSE 255

Room 712 Period M-5 or T-5

ARTHUR LINKSZ, M.D.
New York, N. Y.

Visual Physiology

This will be an informal discussion of some problems of practical visual physiology. The topics will include visual acuity and its correction, binocular vision, muscular balance and imbalance, eyestrain and aniseikonia. The discussion will be held on an elementary basis.

COURSE 256

Room 740 Period M-4 or T-4

RALPH I. LLOYD, M.D.
Brooklyn, N. Y.

Macular Lesions: Differential Diagnosis

The ideal case report should include an adequate history, observation of the lesion over an extended period and, finally, laboratory reports. Unless an eye is removed for pain or imperils the remaining vision or the patient's life, the eye will not reach the laboratory. Under these conditions ophthalmoscopy becomes the main factor in the diagnosis. The value of binocular ophthalmoscopy, serial illustrations in color of various lesions and micro slides of the same or similar cases will be shown. The senile chorioretinal changes through the different stages of simple choroidal atrophy to Coats' disease and disciform degeneration of the macula, with angioid stripes and its similar pathology, will receive preference. The hereditary macular degenerations and other lesions will follow in order of importance, as far as time will allow.

COURSE 257

Room 727 Period T-4

IRVING LUECK
Rochester, N. Y.
BY INVITATION

Practical Optics

A pair of spectacles completely changes the world as seen by the wearer. Hence these spectacles should effect a change in the right amount, and right direction, for the several different situations with which the wearer is occupied in his waking hours.

This course will take up the advantages, limitations, relationships of various types of spectacle lenses available to meet specific applications and some of the more important pitfalls encountered in their use.

COURSE 258

Room 744 Period W-6
or or
Room 734 Period Th-5

JOHN P. MACNIE, M.D.
New York, N. Y.

Head Trauma with Ocular Significance

COURSE 259

Room 752 Period W-6 or Th-6

AUREL E. MANGOLD
New York, N. Y.
BY INVITATION

Proper Fitting of Glasses

This discussion will stress the following subjects:

1. The necessity of analyzing the prescription so that the finished glasses give the maximum of comfort and efficiency
2. Anisometropic prescriptions and effects on reading comfort
3. The importance of individual pupillary distance measurements
4. Lens form and its effect on visual comfort
5. The importance of vertex distance
6. The proper selection of bifocals in cases of hyperopia and myopia
7. Colored lenses
8. Methods of determining causes of patients' complaints with finished glasses
9. Suggested procedure for examination of a pair of glasses

COURSE 260

Room 733 Period W-6 or Th-6

MORTIMER MANN, M.D.
Indianapolis, Ind.

Hypertensive Vascular Changes in the Fundus

To properly interpret the changes produced in the fundus as a result of arterial hypertension, they must be correlated with other clinical and laboratory data. The various ophthalmoscopic findings in essential hypertension, malignant hypertension, toxemia of pregnancy, acute and chronic nephritis will be presented and discussed in relationship to the other manifestations of these diseases. The extent to which observations of the fundi are helpful in prognosis and treatment will also be considered.

COURSE 261

Room 733 Period M-6 or T-6

DON MARSHALL, M.D.
Kalamazoo, Mich.

Lid Infections

Illustrated with kodachrome photographs, acute and chronic infections of the eyelids will be discussed concisely, including therapy. All lid infections are included, but conjunctivitis and lacrimal sac lesions are largely omitted. In addition to the common infections, attention will be given to Parinaud's syndrome, herpes zoster, molluscum, vaccinia, and a few fungus and parasitic infections. The approach throughout will be entirely clinical and practical.

COURSE 262

Room 711 Period M-4 or T-4

MURRAY F. McCASLIN, M.D.
Pittsburgh, Pa.

Ocular Anesthesia

This course will include a brief discussion of the anesthetic agents, curare and hyaluronidase; a review of the anatomy of the lids, orbit, globe, and lacrimal apparatus showing the relationship of landmarks to the injection site for local anesthesia.

Slides and diagrams will be used to illustrate the technics employed for anesthesia of the lids, muscles, intraocular and extraocular surgery, and surgery of the lacrimal apparatus.

Indications for and the use of general anesthesia will be given.

COURSE 263

Room 734 Period W-4 or Th-4

WILLIAM P. McGUIRE, M.D.
Winchester, Va.

Surgical Treatment of Heterophoria and Heterotropia

A brief review of the anatomy and physiology of the extraocular muscles will be given. The preferences of the author, and the reasons therefor, for the use of specific surgical procedures in the various disturbances of ocular motility will be discussed. Technics of these procedures will be demonstrated by slides and motion pictures.

COURSE 264

Room 777 Period W-5 or Th-5

W. HOWARD MORRISON, M.D.
Omaha, Neb.

Office Management of Glaucoma

Glaucoma will be considered from the standpoint of diagnosis and its control as an office procedure. Perimetric studies, provocative tests, with particular reference to phasic variations in ocular tension and slit lamp and gonioscopic examination will be discussed.

Emphasis will be placed on the action and the proper prescribing of miotics. A consideration of office records, tension curves, disc drawings, and environmental difficulties will be undertaken. The psychology of patient-physician relationship will be stressed.

COURSE 265

Room 738 Period W-6 or Th-6

CARROLL R. MULLEN, M.D.
Philadelphia, Pa.

Ophthalmic Emergencies

Treatment of acid and alkali burns of eye and adnexa. Consideration of causes and treatment for sudden loss of vision with special attention to use of anticoagulant medication will be discussed. Value of roentgen studies in ocular injuries with consideration of emergency surgical approach and prognosis. Handling of anterior chamber hemorrhages, iris prolapse and iridodialysis will be studied. Emergency care of lid injuries and trauma to extraocular muscles.

COURSE 266

Room 705 Period M-5

FRANK W. NEWELL, M.D.
Chicago, Ill.

Improving Technic by Means of Rabbit Surgery

The course on animal surgery will consider the following topics:

1. Anatomy of the rabbit eye and orbit
2. Preoperative and postoperative care of animals
3. Anesthesia for various procedures; the use of eyes of animals sacrificed for food
4. Technic of antiglaucoma procedures, cataract extraction, muscle surgery and keratoplasty
5. Legal and ethical considerations

COURSE 267

Room 781 or Room 733 — Period W-5 or Period Th-4

JAMES A. OLSON, M.D.
Detroit, Mich.

Iritis, Iridocyclitis, Keratitis: Differential Diagnosis and Treatment

This course will present a review of the differential diagnostic points in acute, chronic and granulomatous inflammations of the anterior uveal tract and the nonsuppurative inflammatory lesions of the cornea. Attention will be directed to the current methods of therapy, including the use of ACTH, Cortisone and Compound-F. The efficacy of the various methods of administering these agents (intramuscularly, intravenously, subcutaneously, orally, topically or by inhalation), the dosages, and the problems of prolonged treatment and repeated treatment will be discussed. The use of antibiotics in selected cases and other supportive therapeutic measures will be presented. Attention will be directed to the continued importance of careful physical examinations and the subsequent correction of possible causative focal defects or generalized disease processes.

COURSE 268

Room 713 — Period M-4

M. S. OSHER, M.D.
Cincinnati, Ohio

A Diagnostic Approach to Squint Surgery

An accurate diagnosis based on the physiology or actions of the eye muscles is an absolute necessity for good postoperative results in strabismus surgery. An easy understanding of the main actions of the extraocular muscles will facilitate such preoperative diagnosis and will also make simple a quick diagnosis of the various muscle imbalances, particularly vertical imbalances, which may frequently confuse and frighten the ophthalmologist. An accurate diagnosis in itself groups certain muscles as the best probable bets for successful surgery. Tests for motility, convergence, and fixation will help to determine specifically the muscle or muscles to be operated upon.

COURSE 269

Room 743 — Period M-4 or T-4

WILLIAM C. OWENS, M.D.
ELLA M. UHLER OWENS, M.D.
Baltimore, Md.

Retrolental Fibroplasia

The course on retrolental fibroplasia will consider the following topics:

1. Methods of examining the eyes and fundi of premature infants
2. The normal appearance of the eyes and fundi of premature infants
3. The early changes and course of development of retrolental fibroplasia
4. Differential diagnosis of retrolental fibroplasia
5. Management of infants with retrolental fibroplasia
6. Present status of experimental work on retrolental fibroplasia

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COURSE 270

Room 743 — Period M-6 or T-6

FRANK PAYNE, M.D.
New York, N. Y.

Unilateral Visual Failure of Obscure Etiology

This discussion concerns patients having unilateral impairment of vision as the early or outstanding symptom without ocular, medical or neurologic clincal signs which would make the diagnosis fairly obvious. The differential diagnosis considers lesions affecting predominantly one optic nerve. Special emphasis is given to compression lesions since they are too frequently misdiagnosed and consequently progress to produce unilateral blindness and involvement of the other optic nerve. These compression lesions threaten not only the sight but the life of the individual. The examination procedures required in the investigation of such patients are described. Illustrative cases are related. Treatment is briefly mentioned.

COURSE 271

Room 705 — Period W-4 or Th-4

CHARLES A. PERERA, M.D.
New York, N. Y.

Retinal Detachment

The treatment of retinal separation requires an understanding of the etiologic factors and of the pathologic processes involved. Careful preoperative study, adequate surgical technic, and a knowledge of the prognosis as affected by the preopera-

tive and operative findings will improve the surgeon's percentage of successful results. The indications and contraindications for operation and reoperation are also included in this lecture.

COURSE 272

Room 750 Period M-4 or T-4

RAYMOND L. PFEIFFER, M.D.
New York, N. Y.

Differential Diagnosis with Aid of X-ray Findings

Since anatomic and pathologic changes in the orbit can be studied only with the roentgen ray and since in recent years the sphere of usefulness of the roentgen ray has been greatly extended, it is imperative that the ophthalmologist be familiar with its uses and limitations. A vast amount of classified diagnostic material prompting brief discussions of a diversity of subjects will be presented. Exophthalmos, lesions of the chiasmal area, and trauma of the eye and orbit will be considered.

COURSE 273

Room 17 Period T-6

DOHRMANN K. PISCHEL, M.D.
San Francisco, Calif.

Complications of Retinal Detachment Surgery

Abnormal occurrences both in the preoperative and operative period will be discussed as they affect the type of operation, the manner in which it should be performed and the variations that are necessary when various abnormal findings are encountered. Among such findings would be detachments that do not settle out, treatment of detachments without holes, coagulation not showing up in the fundus, poor drainage at the end of the operation, lack of settling out of the retina after good drainage is established and injection of various substances into the eye.

COURSE 274

Room 786 Period M-4

LAWRENCE T. POST, M.D.
St. Louis, Mo.

Difficult Problems in Refraction

Accessory procedures necessary to complicated refractions will be outlined. The refraction of patients with anisometropia, induced phorias, paretic and paralytic complications, accommodative abnormalities, tropias, occupational problems, aphakia, aniseikonia, high myopia, and cylophoria will be considered. Modifications of astigmatic correction, the use of trifocals and contact glasses will be discussed with cases illustrating the management of the refraction in many of these. The increasing exophoria of old age, binocular technic of refraction, and the use of such aids as the stenopaic slit and pinholes will be discussed.

No attempt will be made to give an extended presentation of any of these conditions, but only procedures that have seemed to the speaker especially valuable will be presented.

COURSE 275

Room 726 Period M-4 or T-4

M. HAYWARD POST, M.D.
St. Louis, Mo.

Operating Room Technic

Many factors enter into proper operating room technic, and, without doubt, many an eye has been lost through carelessness in the execution of one or more of these factors.

1. The preparation of the operating room itself
2. The preparation of the patient after arrival at the operating room
3. The preparation of the instruments
4. The maintenance of sterility of the instruments after their preparation is completed
5. The care of the eye and the patient at the conclusion of surgery

These separate factors will be taken up in some detail, with, however, special emphasis on the third and fourth items.

Discussion will be invited.

COURSE 276

Room 711 Period T-5

M. ELLIOTT RANDOLPH, M.D.
Baltimore, Md.

Handling Patients with Advanced Glaucoma

This course will consist of a review of the rationale of various types of medical and/or surgical treatment of cases with advanced glaucoma, with analysis of particular types of advancing field defects and the appropriate medical or surgical treatment.

Advancing glaucoma in the presence of cataract, cataract following glaucoma surgery, glaucoma in the presence of retinal

pathology, and the control of pain in absolute glaucoma will be discussed.

COURSE 277

Room 17 Period M-6

ALGERNON B. REESE, M.D.
New York, N. Y.

Cataract Extraction

Some of the less emphasized features of the operation will be discussed and their importance pointed out. Sutures, forceps, and manner of extraction are excluded and only details, the aggregate of which the author feels makes towards a higher percentage of good results, are included.

COURSE 278

Room 743 Period M-5

CARL J. RUDOLPH, M.D.
South Bend, Ind.

Advantages of General Anesthesia in Ophthalmic Surgery

The advantages of general anesthesia have been long enjoyed by surgeons and patients alike except in intraocular surgery, where the hazards of postanesthetic nausea and vomiting have precluded its use. Since the author first discovered the efficacy of dimenyhydrinate in the prevention and treatment of an induced toxic labyrinthitis caused by the anesthetic agent, it has been made possible to do all types of ophthalmic operations under general anesthesia.

The method of administration is described: thio-pental of sodium intravenously supplemented with nitrous oxide and oxygen by intubation. D-tubo-curarine as an adjunct.

COURSE 279

Room 740 Period M-6 or T-6

WALTER Z. RUNDLES, M.D.
Flint, Mich.

Streak Retinoscopy

The development and use of the streak retinoscope from its invention in 1900 by Hugo Wolff is reviewed. The use of the streak retinoscope is compared with that of the ordinary plane mirror retinoscope and a procedure suitable for routine streak retinoscopy is outlined. Among the advantages of the method are the clarity of the fundus reflex, the ability to measure accurately the refraction in different meridians of the eye individually, the accuracy with which the axis of astigmatism can be determined, the ability to perform retinoscopic examination routinely with contracted pupils and the accurate determination of refraction in the aphakic eye.

COURSE 280

Room 17 Period M-4

RALPH O. RYCHENER, M.D.
Memphis, Tenn.

Common Lesions of the Lids: Method of Treatment

A kodachrome exposition of common diseases of the eyelids encountered in clinical practice, which will include the allergies, oculoglandular syndromes, granulomas, lacrimal affections, malposition of lid borders and ptosis.

Two hundred fifty illustrations of various pathologic states will be presented, with concise remarks about office treatment. An elaboration on treatment will be found in the long abstract received by those in attendance.

COURSE 281

Room 738 Period M-6 or T-6

HAROLD G. SCHEIE, M.D.
Philadelphia, Pa.

Surgery in Congenital Glaucoma

The surgical treatment of juvenile and infantile glaucoma will be discussed. The development of goniopuncture and the results obtained in the treatment of juvenile glaucoma will be presented. The author's method of goniotomy for infantile glaucoma will be illustrated by slides and a motion picture. An analysis of the results obtained from goniotomy, goniopuncture, or a combination of the two will be made.

COURSE 282

Room 705 Period T-5

G. VICTOR SIMPSON, M.D.
Washington, D. C.

Office Management of Convergence Insufficiency and Divergence Excess

This course will consider mainly the medical management of convergence insufficiency and divergence excess after the diagnosis has been made. The criteria upon which a muscle imbalance may be classified into one of these two groups will, however, be discussed.

The items in the management to be stressed will be the proper use of the following measures:

1. Strengthening of convergence movements by pin exercises and loose prism exercises
2. Strengthening of fusion by orthoptics
3. Overcoming suppression as an important point in the management of divergence excess
4. Correction of errors of refraction
5. Use of prisms incorporated in the glasses
6. Elimination of contributing causes such as poor general health, overwork, and emotional factors
7. Elimination of inattention or daydreaming, which allows fusion to be broken up and abnormal divergence to be encouraged

The results of office treatment as measured by the relief of symptoms or change in the position of rest of the eyes will be discussed in this course.

COURSE 283

Room 713 Period T-4 or W-4

ARTHUR A. SINISCAL, M.D.
Rolla, Mo.

Chronic Lid Conditions

A practical course in the recognition, differential diagnosis, and treatment of chronic lid conditions commonly seen in practice. Conditions discussed will be vernal conjunctivitis, folliculosis, trachoma and paratrachoma (swimming-pool) conjunctivitis, oculoglandular conjunctivitis (Parinaud's and tularemic), and chronic catarrhal and pneumococcal conjunctivitis.

Entropion and ectropion (spastic and cicatricial), trichiasis, blepharophimosis, ankyloblepharon, symblepharon and the chronic blepharites will be discussed and illustrated with kodachrome slides.

Treatment with the newer, highly soluble sulfonamides and antibiotics in aqueous buffered ophthalmic solutions will be discussed.

COURSE 284

Room 743 Period W-4 or Th-4

JAMES W. SMITH, M.D.
New York, N. Y.

Practical Points in Office Treatment

Topics considered in the course will be consultation room principles, aids in diagnosis, treatment and technics used in examinations and refractions. From personal office experiences the lecturer will discuss some important diagnostic and therapeutic pitfalls in ophthalmic practice that should be recognized and avoided.

COURSE 285

Room 706 Period M-4

EDMUND B. SPAETH, M.D.
Philadelphia, Pa.

Surgical Diseases of the Lacrimal Gland and Lacrimal Sac

The lecturer will discuss the surgical therapy of lacrimal cystectomy and cystorhinostomy and the indications and the contraindications for each. In addition, such conditions as tumors of the lacrimal gland, intractable epiphora, lacerations of the canaliculi, traumatic sectioning of the lacrimonasal duct, and malignancy of the prelacrimal regions will be discussed.

COURSE 286

Room 706 Period T-4

EDMUND B. SPAETH, M.D.
Philadelphia, Pa.

Plastic Surgery: Practical Aspects

This course has as its purpose a consideration of the end results one should logically expect from the surgical correction of certain plastic defects of the lids and the orbit. Many of these defects can be corrected to such a degree that the postoperative condition is practically normal. In others the improvement possible cannot be so complete. A small number of plastic conditions are best treated by procedures other than surgery. It is wise, for all concerned, that these facts be recognized.

Entropion and ectropion, traumatic ptosis, congenital colobomas of the upper and lower lids, major plastic defects of the lids, symblepharon, and the contracted socket will be considered in more or less detail. Case reports, as presented by lantern slides, and the blackboard, for sketching, will be used for the presentation.

COURSE 287

Room 781 Period T-6 or W-6 or Th-6

ELLIS H. STEFFENSEN, M.D.
Detroit, Mich.

The Use of ACTH and Cortisone in Ophthalmology: Contraindications, Complications and Clinical Value

Ophthalmology has witnessed a widespread clinical trial of adrenocorticotropic hormone (ACTH) and cortisone during the past two and one-half years, and the ac-

cumulated results of many investigators now more than justify the common use of these drugs in a variety of ocular diseases.

Indications for the use of ACTH and of cortisone in both parenteral and topical administration will be discussed. Currently accepted therapeutic programs for the treatment of acute and chronic lesions will be outlined, together with the precautionary measures necessary for the protection of the patient from possible adverse pharmacologic side effects. The physiologic basis for the use of these drugs in the treatment of various ocular lesions will be briefly reviewed.

COURSE 288

Room 750 Period W-6 or Th-6

RUSSELL L. STIMSON
Los Angeles, Calif.
BY INVITATION

Lenses for the Aphakic

A simplified procedure by which cataract lens prescriptions are interpreted into best lens forms will be described. The salient optical problems of the aphakic eye and its correcting lens will be briefly reviewed. The fitting method will be evaluated by comparison of the summarized results of numerous ophthalmologists covering a three year period.

COURSE 290

Room 14 Period M-5

KENNETH C. SWAN, M.D.
Portland, Ore.

Value of New Drugs in Ophthalmology

Recently a number of enzyme-like compounds have been purified and applied clinically. Some new anesthetic agents, analgesics, and sedatives have been receiving clinical trial. Also the role of cortisone and related hormones in ophthalmic therapy has been clarified. Some new principles of applying older drugs have been recognized. A contemporary evaluation of these therapeutic developments will be presented.

COURSE 291

Room 743 Period T-5 or W-5

EDWIN FORBES TAIT, M.D.
Norristown, Pa.

Practical Methods in the Correction of Accommodation and Convergence Abnormalities

Accommodation and convergence are considered in terms of the stimuli which produce them as responses.

Most accommodation and binocular vision abnormalities are the result of deficiencies in those reflexes. On this basis, therefore, a clinically useful classification of abnormalities can be established, affording a rational basis for orthoptic, refractive, or surgical corrective procedures.

Based upon the above, practical methods for the correction or relief of accommodation and convergence abnormalities will be presented.

COURSE 292

Room 750 Period W-4 or Th-4

FREDERICK H. THEODORE, M.D.
New York, N. Y.

Ocular Therapeutics

The course will offer a practical evaluation of old and new drugs used in the treatment of external diseases of the eye. The actions of the drugs themselves and their therapeutic applications, as well as sensitizations and irritations arising from their use, will be considered. The choice of appropriate vehicles and other problems relating to the prescribing of drugs will be discussed. Kodachromes will be used to demonstrate clinical cases and methods of treatment.

COURSE 293

Room 728 Period W-5 or Th-5

ROBERT H. TRUEMAN, M.D.
Philadelphia, Pa.

Early Indications and Differential Diagnosis of Cerebral Lesions

The purpose of this presentation is to review the early ocular signs and symptoms of intracranial lesions. While it is possible to localize intracranial lesions by only the ocular signs, the addition of neurologic signs and symptoms increases the localizing possibilities. It is for this reason that the more important nonocular manifestations are included.

It is felt that the most logical approach to the subject is in the various anatomic locations in which the lesions may occur. In this way, by means of slides, brief, pertinent reviews of the anatomy of the brain can be used to illustrate the clinical manifestations of the lesions.

COURSE 294

Room 740 Period M-5 or T-5

CLARENCE A. VEASEY, JR., M.D.
Spokane, Wash.

Refraction Difficulties

This course assumes that glasses have been prescribed and the patient is still unhappy. Usual and unusual sources of discomfort will be considered, including adjustment requirements for distance and near, decentration of plus lenses, disturbances of accommodation-convergence relationship and necessity for tilting, as well as the necessity for changing optical center when a lens is tilted and allowing for induced increased sphere and cylinder.

Measurement and importance of vertex distance will be discussed. When vertex distance in finished glasses differs from that of the trial frames, a change in strength of both sphere and cylinder is necessary. Calculation of prismatic effect of spheres and cylinders of various axes will be demonstrated, as well as calculation of hyperphoria induced by anisometropia and remedies thereof, including compensated segments, bicentric grinding and prisms. Special problems of multifocal lenses and aphakia will be presented. All calculations will involve arithmetic only and inspection of tables. Abstracts cannot include illustrations. The tables essential to applying the principles elucidated in this course can be obtained in the author's instruction manual, "Refraction Difficulties," published by the Academy.

COURSE 296

Room 17 Period T-5

FRANK B. WALSH, M.D.
Baltimore, Md.

Interesting Neuro-ophthalmologic Cases: Difficult Clues in Diagnosis

A collection of cases exemplifying lesions of the optic nerves and chiasm. This has been selected as a topic on the basis that each case illustrates an important diagnostic consideration.

COURSE 297

Room 712 Period M-4 or T-4

HAROLD F. WHALMAN, M.D.
Los Angeles, Calif.

Minor Surgical Procedures of the Eye

This presentation comprises the instructor's experience in several ophthalmic operations such as those for papilloma, xanthoma, hemangioma, melanosis, chalazion, pinguecula, pterygium, mucous membrane transplant, accessory lacrimal gland, and lacrimal excretory apparatus.

Securing good local anesthesia easily and safely is as essential to success as is meticulous attention to minute details of procedure, and it will be the endeavor of the instructor to present these practical considerations.

COURSE 298

Room 17 Period W-4

MEYER WIENER, M.D.
Coronado, Calif.

Minor Lid Surgery

In the short time allotted, it is impossible to cover the entire field of minor lid surgery. Therefore, certain methods or procedures have been selected for the correction of lid defects, growths, or injuries which have been found personally advantageous. Lantern slide and blackboard demonstrations will be used to help clarify the advocated procedures. Attempt will be made to give reasons why a certain method is advised and how, or the manner in which the effect is accomplished.

COURSE 299

Room 740 Period W-6 or Th-6

WILLIAM A. MANN, M.D.
Chicago, Ill.

Refraction and Lens for the Cataract Patient

Problems of refraction of the patient with early cataract. Correction of aphakia. Methods of objective and subjective testing. Precautions in ordering lenses. Analysis of unsatifactory visual results with finished glasses. Types of cataract lenses. Methods of securing single binocular vision in bilateral and monocular aphakia. Catmin lenses, contact lenses.

HOME STUDY COURSES DISCUSSION PERIODS

The Home Study Courses Discussion Periods are based on the Home Study Courses lesson material for each particular subject. Those attending the discussion periods will be provided at the time of the meeting with a copy of the questions prepared for the particular subject. The members of the faculty will offer opinions on

the clinical application of the questions and will encourage an exchange of ideas.

These discussion periods will be profitable to physicians who desire to review basic subjects and obtain their up-to-date clinical application. They are intended for physicians who have completed or who are taking the Home Study Course in Ophthalmology sponsored by the Academy. Members of the Home Study Courses faculty and others outstanding in their respective fields will be present to answer questions or discuss problems which have arisen from the reading assignments or the monthly questions sent to registrants in the Home Study Courses.

Tickets for each discussion period may be purchased individually at $2.00 per hour. The students are urged to bring their problems to the lecture room or to write in advance to one of the faculty members.

HOME STUDY A

Room 782 Period M-4

Embryology, Anatomy and Neuro-Anatomy

Raymond E. Meek, M.D.
New York, N. Y.

John W. Henderson, M.D.
Ann Arbor, Mich.

HOME STUDY B

Room 782 Period M-5

Physiology

Hendrie W. Grant, M.D.
St. Paul, Minn.

George P. Guibor, M.D.
Chicago, Ill.

HOME STUDY C

Room 782 Period M-6

Optics and Principles of Biomicroscopy

Arthur Linksz, M.D.
New York, N. Y.

Frederick C. Cordes, M.D.
San Francisco, Calif.

HOME STUDY D

Room 782 Period T-4

Refraction

Daniel Snydacker, M.D.
Chicago, Ill.

Frank W. Newell, M.D.
Chicago, Ill.

Thomas D. Allen, M.D.
Chicago, Ill.

HOME STUDY E

Room 782 Period T-5

External Diseases, Bacteriology and Virology

James H. Allen, M.D.
New Orleans, La.

Capt. Samuel N. Key, Jr. (MC)
Keesler Air Force Base, Miss.

HOME STUDY F

Room 782 Period T-6

Perimetry and Neuro-Ophthalmology

Placidus J. Leinfelder, M.D.
Iowa City, Iowa

Cecil W. Lepard, M.D.
Detroit, Mich.

HOME STUDY G

Room 782 Period W-4

Extraocular Muscles

Paul T. McAlpine, M.D.
Summit, N. J.

Hunter H. Romaine, M.D.
New York, N. Y.

Conrad Berens, M.D.
New York, N. Y.

COURSE H

Room 782 Period W-5

Ophthalmoscopy and Medical Ophthalmology

John P. Macnie, M.D.
New York, N. Y.

William Banks Anderson, M.D.
Durham, N. C.

COURSE I

Room 782 Period W-6

Pathology

Michael J. Hogan, M.D.
San Francisco, Calif.

John S. McGavic, M.D.
Bryn Mawr, Pa.

Georgiana D. Theobald, M.D.
Oak Park, Ill.

COURSE J

Room 782 Period Th-4

Pharmacology and Therapeutics

Kenneth C. Swan, M.D.
Portland, Ore.

Alfred J. Kreft, M.D.
Portland, Ore.

CONTINUOUS COURSES — OTOLARYNGOLOGY

COURSE 101

Room 752 Periods T-1, T-2

FRANZ ALTMANN, M.D.
JULES WALTNER, M.D.
New York, N. Y.

Histopathology of the Ear with Practical Applications—Part I

A two-period course—$4.00

COURSE 102

Room 754 Periods W-1, W-2

FRANZ ALTMANN, M.D.
JULES WALTNER, M.D.
New York, N. Y.

Histopathology of the Ear with Practical Applications—Part II

A two-period course—$4.00

Courses 101 and 102 are limited to individuals who have taken the Academy course on "Histology of the Ear" or its equivalent. They will be comprised of low and high power study of sections of the temporal bone, covering the common pathologic conditions there encountered. Particular attention will be paid to otosclerosis and related bone diseases as well as to inflammatory and neoplastic diseases. There will be a lantern slide demonstration followed by study of the actual specimens.

COURSE 103

Room 744 Periods T-1, T-2

COURSE 104

Room 744 Periods W-1, W-2

WILLIAM T. K. BRYAN, M.D.
and
MARIAN P. BRYAN
BY INVITATION
St. Louis, Mo.

Cytologic Pathology of Ear, Nose and Throat Diseases

A two-period course—$4.00

This two-hour course is being presented twice, as Course 103 on Tuesday and as Course 104 on Wednesday. **Please select course number according to day on which you wish to attend.**

What changes occur within the individual tissue cells which compose many of our pathologic patterns? The exfoliative cytology of selected cases will be correlated with pathologic tissue sections from the same cases. Malignant cells of various types, as well as radiation changes in individual cells from our tumor collection, will be shown. Intracellular morphology of columnar ciliated epithelial cells in acute upper respiratory infections and their significance thus far as seen with the Papanicolaou stain will be included. Emphasis will be given to aural cytology with reference to the differential diagnosis by smear of acute and chronic middle ear suppurations. All points of cytology will be illustrated with kodachrome slides taken at high magnification. A resume of adaptable staining technics for exudates in ear, nose and throat will be given. Such patterns of cytologic pathology correlated with clinical symptoms present an awareness of the ever-changing processes of disease. Their use will aid in the diagnosis of difficult cases.

COURSE 105

Room 786 Periods T-1, T-2

COURSE 106

Room 786 Periods Th-2, Th-3

JOHN J. CONLEY, M.D.
New York, N. Y.

Cancer of the Head and Neck: Diagnosis and Treatment

A two-period course—$4.00

This two-hour course is being presented twice, as Course 105 on Tuesday and as Course 106 on Thursday. **Please select course number according to day on which you wish to attend.**

The course includes an analysis of cancers involving all the major systems of the head and neck area. This includes the various types of malignant tumors involving the epithelium, the sinuses, the ear, the lips, the tongue, the buccal mucosa, the palate, the nasopharynx, floor of the mouth, hypopharynx, parotid gland, cervical lymphatics, thyroid gland and cervical esophagus (larynx intentionally omitted because of its inclusion in other courses). The diagnosis of these various types of malignant tumors will be discussed in the pertinent factors pertaining to the history, physical examination, x-ray and chemical tests and various biopsy technics relative to each case.

A discussion of treatment will include the curative efforts embracing various

types of surgery and irradiation and combinations of these two technics. It will also include the worth-while palliative efforts in the management of incurable cancer by surgery, irradiation or combinations of these technics.

COURSE 107

Room 752 Periods W-1, W-2

JOHN MARQUIS CONVERSE, M.D.

New York, N. Y.

Principles of Nasal Plastic Surgery—Basic

A two-period course—$4.00

This course will review the main steps in the typical corrective nasal plastic operation. The course will be illustrated by slides and motion pictures.

COURSE 108

Room 752 Periods Th-2, Th-3

JOHN MARQUIS CONVERSE, M.D.

New York, N. Y.

Principles of Nasal Plastic Surgery—Advanced

A two-period course—$4.00

This course is more advanced than the preceding one and deals, in addition to the main steps in the typical corrective nasal plastic operation, with conditions such as nasal deviations and saddle nose.

COURSE 109

Room 736 Periods T-2, T-3

COURSE 110

Room 736 Periods W-2, W-3

FREDERICK A. FIGI, M.D.

Rochester, Minn.

Lesions of the Mouth

A two-period course—$4.00

This two-hour course is being presented twice, as Course 109 on Tuesday and as Course 110 on Wednesday. **Please select course number according to day on which you wish to attend.**

Lesions of the mouth usually occur as a purely local process; however, they may be a manifestation of some systemic disturbance. A great variety of pathologic conditions, including inflammatory, metabolic and neoplastic lesions, occur in the oral cavity. Those of greatest clinical importance and of especial interest for other reasons will be presented for consideration by means of lantern slides.

COURSE 111

Room 754 Periods T-1, T-2

EDMUND P. FOWLER, JR., M.D.

and

FERNAND MONTREUIL, M.D.

BY INVITATION

New York, N. Y.

Histology of the Ear—Vertical Sections

A two-period course—$4.00

COURSE 112

Room 754 Periods Th-1, Th-2

EDMUND P. FOWLER, JR., M.D.

and

FERNAND MONTREUIL, M.D.

BY INVITATION

New York, N. Y.

Histology of the Ear—Horizontal Sections

A two-period course—$4.00

Low and high power study of horizontal and vertical sections through the temporal bone at various critical levels.

The aim of these courses is to teach the participants enough cross-section anatomy in the temporal bone so that when they see medical illustrations of histologic material they will be oriented. Each participant will study selected serial sections of the temporal bone, color and label tracings so as to identify the important structures. High and low power microscopes will be available for each student, but it is requested that each participant provide himself with a box of hard colored pencils.

COURSE 113

Room 731 Periods T-1, T-2

COURSE 114

Room 731 Periods Th-1, Th-2

FRENCH K. HANSEL, M.D.

St. Louis, Mo.

Allergy in Otolaryngology: Practical Procedures in Diagnosis and Treatment

A two-period course—$4.00

This two-hour course is being presented two times, as Course 113 on Tuesday and as Course 114 on Thursday. **Please select course number according to day on which you wish to attend.**

This course is designed as a practical approach to the problems of diagnosis and treatment of allergy as related to otolaryngology. Particular emphasis will be placed upon history taking and possible etiologic factors. The technic and interpretation of

skin tests will be demonstrated. The microscopic study of secretions and pollen grains will be included. Finally, the methods of allergic management, with particular reference to the treatment of food, inhalant and pollen allergies, will be presented.

COURSE 115

Room 752 and Room 744 — Period T-3, W-3 and Period Th-2

CHEVALIER L. JACKSON, M.D.
Philadelphia, Pa.

Problems of Technic in Peroral Endoscopy

A three-period course—**$6.00**

Consideration of location and character of room suitable for endoscopic clinic. Lighting and other facilities required. Types of table and other furniture. Instrumentarium for various kinds of endoscopic clinics. Anesthesia. Demonstration of position of patients for direct laryngoscopy, bronchoscopy, esophagoscopy and gastroscopy, illustrated by means of motion picture films. Technic of introduction of instrument with especial attention to the principal difficulties and ways of overcoming them. Technic of use of forceps for bronchoscopic biopsy and removal of benign tumors. Technic of electrocoagulation. Technic of catheterization of segmental bronchi. Solution of typical mechanical problems of foreign body extraction.

COURSE 116

Room 738 — Periods T-1, T-2

V. H. KAZANJIAN, M.D.
Boston, Mass.

JOHN MARQUIS CONVERSE, M.D.
New York, N. Y.

EDGAR M. HOLMES, M.D.
Boston, Mass.

Traumatic Injuries of the Face

A two-period course—$4.00

The symposium on early treatment of facial injuries, held during the first period, will cover:

1. Emergency measures consisting of the control of primary hemorrhage, traumatic shock, and other necessary first aid measures
2. Local examination of lacerations and fractures of the face plus general physical examination
3. Early treatment of soft tissues, primary, and late primary suturing of lacerations, and the immobilization of fractures with special reference to fractures of the nasal, zygomatic and maxillary bones.

The second period will be comprised of a symposium on treatment of deformities of the face of traumatic origin, including:

1. Discussion of some general principles of reconstructive surgery of the face
2. Repair of deformities involving soft tissues only
   a. Conspicuous scars without apparent loss of tissue
   b. Defects with definite loss of tissue requiring use of pedicle or skin grafts
   c. Treatment of special areas such as the eyelids, eyebrows, and stenosis of the nasal passages
3. Repair of deformities of bony structure of the face
   a. Defects of the nose
   b. Defects of the zygoma
   c. The relative value of cartilage and bone as transplants.

Following presentation of these topics, ample time will be allowed for open discussion which, in the past, has proved most valuable.

COURSE 117

Room 722 — Periods T-2, T-3

COURSE 118

Room 722 — Periods W-2, W-3

COURSE 119

Room 722 — Periods Th-2, Th-3

JULIUS LEMPERT, M.D.
New York, N. Y.

Practical Clinical Means for Diagnosing the Type and Evaluating the Degree of Hearing Function in a Given Ear

A two-period course—$4.00

This two-hour course is being presented three times, as Course 117 on Tuesday, as Course 118 on Wednesday, and as Course 119 on Thursday. **Please select course number according to day on which you wish to attend.**

1. How to elicit a correct history of a hearing loss, which could lead to the correct diagnosis.
2. How to evaluate the history when correlated to the physical findings in the ear.
3. How to test for existing pure unadulterated physiologic hearing function in a given ear.
4. How to differentiate between a middle

ear, inner ear or combined middle and inner ear hearing function loss.
5. How to determine whether a hearing loss is correctable.
6. How to determine the best way of improving the hearing in a particular ear and under circumstances which could best serve the particular patient.

COURSE 120
Room 738 Periods W-2, W-3
COURSE 121
Room 738 Periods Th-2, Th-3

JOHN R. LINDSAY, M.D.
Chicago, Ill.

Vertigo: Differential Diagnosis and Treatment

A two-period course—$4.00

This two-hour course is being presented twice, as Course 120 on Wednesday and as Course 121 on Thursday. **Please select course number accoring to day on which you wish to attend.**

Vertigo caused by a disturbance of the inner ear or the vestibular pathways is differentiated from dizziness of other origins.

This course reviews in detail the essentials of an adequate history and the routine ear, nose and throat and functional examinations, and discusses the interpretations.

A practical classification of vertigo is presented with demonstration by case history of the various causes of vertigo within each group and the appropriate management.

COURSE 122
Room 727 Periods T-1, T-2
COURSE 123
Room 727 Periods W-2, W-3

JACQUES W. MALINIAC, M.D.
New York, N. Y.

Pitfalls in Maxillofacial Plastic Surgery: Prevention and Correction

A two-period course—$4.00

This two-hour course is being presented twice, as Course 122 on Tuesday and as Course 123 on Wednesday. **Please select course number according to day on which you wish to attend.**

The purpose of this course is to review the early and late sequelae which occur in early and late repair of some maxillofacial deformities. The majority of these secondary deformities can and should be prevented.

1. Congenital Nasal Deformities
   a. Complications due to improper planning and artistic considerations
   b. Sequelae due to faulty surgical technic
2. Post-traumatic Nasal Deformities
   a. Sequelae in early repair
   b. Sequelae in reconstruction of depressed and deviated deformities
   c. Complications due to the use of supporting grafts
   d. Skin grafts and flaps required in secondary nasal deformities
3. Subtotal and Total Nasal Losses
   a. Subtotal losses following corrective rhinoplasty and their repair
   b. Losses due to injury and disease—reason for failure
4. Sequelae in Soft Tissue Repair
   a. The problem of scars—their prevention and treatment
   b. The proper choice of free grafts and flaps for facial repair
5. Inadequateness in Reconstruction of Ear
   a. Malpositions
   b. Partial or total loss

COURSE 124
Room 731 Periods W-1, W-2

RICHARD E. MARCUS, M.D.
Chicago, Ill.
and
JOHN E. BORDLEY, M.D.
Baltimore, Md.
BY INVITATION
HELMER R. MYKLEBUST, Ph.D.
Evanston, Ill.
BY INVITATION
JACQELINE KEASTER
Iowa City, Iowa
BY INVITATION

Hearing Problems in Children

A two-period course—$4.00

To be presented are (a) methods of diagnosis in the preschool and school-age child, (b) technics available for differential diagnosis, (c) the otolaryngologist and school hearing surveys, including methods of screening, interpretation of results, and treatment, (d) rehabilitative procedures: use of hearing aids, auditory training, speech and lip reading. The material will be presented in symposium form, stressing highlights and practical detail. Each member of the symposium panel will discuss his subject, following which the audience will be invited to participate and questions about special problems will be answered.

COURSE 125

Room 733 Periods W-2, W-3

PAUL J. MOSES, M.D.
San Francisco, Calif.
BY INVITATION

Speech and Voice Therapy in Otolaryngology

A two-period course—$4.00

Every ear, nose and throat specialist sees in his office a large number of patients with voice and speech problems. Notably, hoarseness without visible organic change is a frequent occurrence. The field of the diseases of speech and of the speaking and singing voice, including the functional and psychosomatic aspects of diagnosis and treatment, should be part of the training of the laryngologist. It is a medical field which should not be handled by the voice and speech teacher without supervision by the otolaryngologist.

This course will concentrate on practical office procedures for the analysis and treatment of disturbance of voice and speech. Acoustic diagnosis will be illustrated by phonograph records. The program comprehends the organic problems of laryngeal diseases: nodes, contact ulcers, paralysis of vocal cords, endocrine changes (mutation and its pathology), re-education of voice after laryngectomy.

Functional diseases include "professional" voice disturbances, vocal neurosis, persistent falsetto voice, pathology of the singing voice and the vocal pathology of the school child. A voice hygiene program for schools will be outlined.

Pathology and therapy of speech comprises: speech development, hearing mutism, functional speech disorders like lisping, stuttering, soft palate problems (paralysis, cleft palate) and aphasia.

This course will emphasize the strictly practical approach to these problems.

COURSE 126

Room 724 Periods W-2, W-3

COURSE 127

Room 724 Periods Th-1, Th-2

WERNER MUELLER, M.D.
Boston, Mass.

Practical Audiometry

A two-period course—$4.00

This two-hour course is being presented twice, as Course 126 on Wednesday and as Course 127 on Thursday. **Please select course number according to day on which you wish to attend.**

1. Practical versus scientific audiometry.
2. What do we mean by "accuracy"?
3. Do we need soundproof rooms?
4. The "inaccurate" and "unscientific" voice test and why it is important to us.
5. Don't throw away your tuning forks yet.
6. Cycles and decibels made easy.
7. The audiometer and how to use it. What it tells us and what it doesn't.
8. The interpretation of audiograms.

COURSE 128

Room 733 Periods T-2, T-3

COURSE 129

Room 733 Periods Th-2, Th-3

HENRY B. ORTON, M.D.
Newark, N. J.

Applied Surgery of the Neck

A two-period course—$4.00

This two-hour course is being presented twice, as Course 128 on Tuesday and as Course 129 on Thursday. **Please select course number according to day on which you wish to attend.**

The course will include the procedures of neck surgery, reviewing very briefly the fascial planes, surgical landmarks, and the important anatomic levels in the neck, before taking up the following surgical procedures:

Surgical approaches to deep infections of the neck; resection of the submaxillary gland; block dissection of the neck; thyroglossal duct cysts; branchial cysts and fistulas; cystic hygroma colli; tracheotomy; thyrotomy; laryngectomy; laryngo-pharyngectomy and plastic repair; lateral transthyroid pharyngotomy; arytenoidectomy; cervical esophagotomy and cervical mediastinotomy; pharyngeal diverticulum; phrenectomy; approach to roots of brachial plexus; operations on the thyroids; traumatic wounds of the neck; laryngostomy; and ligations.

Due to the large number of surgical procedures to be covered within the allotted time, the history, symptoms, and diagnosis of the various conditions will be omitted.

These operations will be shown by means of colored motion pictures and colored lantern slides.

COURSE 130

Room 732 Periods T-1, T-2

COURSE 131

Room 732 Periods Th-2, Th-3

LYLE M. SELLERS, M.D.
Dallas, Texas

Equilibration: Balance, Imbalance and Vertigo

A two-period course—$4.00

This two-hour course is being presented twice, as Course 130 on Tuesday and as Course 131 on Thursday. **Please select course number according to day on which you wish to attend.**

Implementation of the otolaryngologist in contending successfully with a troublesome clinical problem is the object of this course. Much confusion exists concerning the practical management of the multiphased clinical entity of vertigo. Various regimens of treatment have been advanced but many of these actually represent the clinical management of but a single separate phase.

By presenting clearly the entire physiologic process of equilibration with its normal state of balance, a clear-cut understanding of the various disturbances leading to imbalance and vertigo is made possible.

By still projections and by motion pictures the clinical tests are demonstrated and explained so that they may be performed readily by the otolaryngologist in his office. By illustrative actual case reports these then are applied to both the simpler and the more difficult problems. The solution of these problems and the appropriate treatment with the results obtained are then given.

COURSE 132

Room 734 Periods T-2, T-3

COURSE 133

Room 734 Periods Th-2, Th-3

HERMAN SEMENOV, M.D.
Beverly Hills, Calif.

Current Therapy in Relation to the Applied Pathology of the Ear, Nose and Throat

A two-period course—$4.00

This two-hour course is being presented twice, as Course 132 on Tuesday and as Course 133 on Thursday. **Please select course number according to day on which you wish to attend.**

This course in applied pathology may be regarded as a clinical-pathological conference in which sufficient time is allowed for questions and discussion, not only of the pathology of each disease, but also various methods of treating it. The salient features of the pathology will be clearly demonstrated by means of kodachrome lantern slides, and the important histology of the common, everyday lesions of the ear, nose and throat will be shown in detail. In the discussion that follows each demonstration, diagnostic and therapeutic comments pertaining to the choice of medical and/or surgical management will be entertained. The main purpose of this course, therefore, is a practical one: (1) to bring our knowledge of pathology up to date, and (2) to provide a solid foundation for time honored procedures as well as newer methods of therapy. The agenda includes a wide variety of topics: rhinitis and sinusitis, infection and allergy, cytologic diagnosis of smears, tonsils and adenoids, nasopharyngeal lymphoid tissue, acute and chronic otitis media, otosclerosis and deafness, laryngeal nodes, the pathology of benign and malignant tumors, focal infection and collagen disease. In addition mention will be made of the indications for or against surgery, irradiation, cortisone, varidase, tryptar, and other pertinent clinical consideration.

COURSE 134

Room 777 Period T-1
Room 734 Period W-3
Room 711 Period Th-1

GABRIEL TUCKER, M.D.
JOSEPH P. ATKINS, M.D.
and
WILLIAM A. LELL, M.D.
BY INVITATION
Philadelphia, Pa.

Esophageal Diseases and Foreign Bodies: Diagnosis and Treatment

A three-period course—$6.00

COURSE 135

Room 776 Period T-3
Room 712 Period W-2
Room 711 Period Th-2

GABRIEL TUCKER, M.D.
JOSEPH P. ATKINS, M.D.
and
WILLIAM A. LELL, M.D.
BY INVITATION
Philadelphia, Pa.

Laryngeal Surgery

A three-period course—$6.00

COURSE 136
Room 726 Periods W-1, W-2

COURSE 137
Room 716 Periods Th-2, Th-3

FRANK TURCHIK, M.D.
Bridgeport, Conn.

Surgical Treatment of Nasal Obstruction

A two-period course—$4.00

This two-hour course is being presented twice, as Course 136 on Wednesday and as Course 137 on Thursday. **Please select course number according to day on which you wish to attend.**

Treatment of nasal obstruction is no longer simple submucous resection. When proper consideration is given to the extraseptal factors in the nasal pyramid in evaluating good physiologic nasal breathing, one must conclude that only "rhinoplastic septoplasty" can achieve satisfactory restoration of function.

In the preoperative management, the role of pathologic processes local in nature plus general and systemic diseases that affect the nasal mucosa must be properly evaluated and the extent of their contribution to the nasal obstruction accurately estimated. Diseases that prevent or delay normal healing must be recognized preoperatively and controlled before surgery is committed.

The surgery is dependent on familiarity with the anatomy of the external nasal pyramid and an understanding of nasal physiology, which permits an accurate diagnosis of nasal obstruction.

Corrective surgical technics and methods for preventing postoperative sequelae will be presented with the aid of lantern slides.

Septal surgery in children is indicated and justifiable if conservatism is practiced. Prophylaxis embraces proper examination and treatment in the delivery room and early and proper treatment of nasal trauma.

In summary, the diagnosis in nasal obstruction is all-important, the surgery simple, and results most gratifying to both physician and patient.

INDIVIDUAL COURSES — OTOLARYNGOLOGY

COURSE 401
Room 712 Period T-1 or W-1

LUCIAN W. ALEXANDER, M.D.
New Orleans, La.

TheAdenoid Operation and Its Relation to the Auditory Apparatus

There is no operation in surgery so badly done in such a large percentage of cases as the adenoid operation. A discussion of the many technics, failure in results, postoperative treatment to avoid middle ear infections, the management of adenoid remnants and case histories will be presented.

COURSE 402
Room 712 Period T-2 or Th-2

REA E. ASHLEY, M.D.
San Francisco, Calif.

Dust and Other Perennial Inhalant Therapy in Respiratory Allergy

1. Discussion of the importance of molds, house dust and industrial dusts as etiologic factors in nasal and other respiratory allergy.
2. Short-cut methods of diagnosis of dust allergy.
3. Treatment of dust allergy including prophylaxis, value of drugs, hyposensitization by sublingual absorption and by injection.
4. Practical aspects. Where to obtain materials. How to make dilutions. How to determine the size of initial doses. How to vary the dose to obtain the desired effect.
5. Results. Reasons for successes and failures.

COURSE 403
Room 705 Period T-1 or W-1

SHIRLEY H. BARON, M.D.
San Francisco, Calif.

Modified Radical Mastoidectomy: Preservation of the Cholesteatoma Matrix

The modified radical mastoidectomy has not been given the consideration it deserves in the surgical treatment of the chronic otorrheas. It is felt that in many instances, radical mastoid surgery has been done on patients where the modified radical operation would have sufficed.

To support this view there will be out-

lined: (1) the purpose of the modified radical mastoidectomy; (2) the indications; (3) certain situations which will determine the selection of the modified radical operation. The technic of surgery, both endaural and postauricular, will be reviewed.

The management of the cholesteatoma matrix, about which there is much confusion and difference of opinion, will be discussed. The genesis and classification of cholesteatoma will be outlined and indications for the preservation of the cholesteatoma matrix will be presented.

The course will be illustrated by slides and kodachrome motion pictures.

COURSE 404

Room 705 Period T-2 or W-2

OSCAR J. BECKER, M.D.
Chicago, Ill.

Plastic Surgery of Protruding Ears

An original technic of correcting protruding ears is to be presented. This method has proved highly satisfactory in reproducing the normal contours of the auricle and in setting the ear back to the head at the proper angle.

The anatomy of the external ear, the etiology and pathology of protruding ears, and a historical survey of operative procedures will be included in the discussion.

COURSE 405

Room 711 Period T-3 or W-3

RICHARD J. BELLUCCI, M.D.
New York, N. Y.

Principles of Submucous Resection of the Nasal Septum

Surgical anatomy of the nasal septum and framework of the external nose will be reviewed.

A simplified technic for the surgical correction of the deviated nasal septum will be presented as taught at the Manhattan Eye, Ear, and Throat Hospital.

Surgical management of the anterior deviated nasal septum with obstruction of the nares will be presented.

The relative importance of the septum in plastic surgery of the nose will be discussed.

COURSE 406

Room 706 Period T-1 or Th-1

GUY L. BOYDEN, M.D.
Portland, Ore.

Chronic Frontal Sinus Surgery

A method in which a mucoperiosteal flap is used to make a new opening into the frontal sinus without the use of tantalum or any other foreign body will be discussed.

Slides, a motion picture, and specimen will be used to demonstrate the procedure.

COURSE 407

Room 706 Period T-2 or W-2

NORTON CANFIELD, M.D.
New Haven, Conn.

Audiology for the Practicing Otologist

Based on the experience of examination, diagnosis, treatment and advice for several thousand patients with hearing loss who have sought my attention as an otologist during the past twelve years, a definite management pattern has evolved which provides these patients with a combination of services. Believing that those with impaired hearing can best be managed and advised by an otologist through all phases of their rehabilitation, the material to be presented will emphasize those features of professional attention which are so eagerly sought after by those with impaired hearing. Special attention will be given to those cases for whom the otologist usually is unable to provide specific therapy for improvement of hearing acuity. The services available to the deafened outside of the otologist's office will be discussed. Testing apparatus, including the speech audiometer, and the cooperative help of a trained assistant, will indicate the possibilities for expansion of the otologist's work in the future.

COURSE 408

Room 722 Period W-1 or Th-1

RAYMOND CARHART, Ph.D.
Evanston, Ill.
BY INVITATION

The Audiogram in Otologic Diagnosis

The course emphasizes aspects of pure tone audiometry which are particularly important in its applicaton to diagnostic problems. Methods for clinical calibration of both the air conduction and bone conduction systems are discussed. Particular

emphasis is given to the interpretations allowed by combined consideration of air and bone measurements. This emphasis includes review of both the basic differentiations which are possible and the audiometric configurations associated with various pathologic conditions. The audiometric patterns characterizing otosclerosis are analyzed to illustrate the problems and methods in estimating cochlear reserve.

COURSE 409

Room 712 Period T-3 or W-3

SAMUEL COHEN, M.D.
Philadelphia, Pa.

Deviated Noses and Their Correction

Deviated noses are classified into four groups: (1) cartilaginous; (2) bony; (3) combined bony and cartilaginous; and (4) the dislocated septal cartilage.

Preliminary to all corrective procedures, the nasal skin is elevated and the septal cartilage is cut from the columella.

The bony deviation is corrected by removing a bony triangle from the dorsum. The nasal processes are cut and fractured at equal or unequal distances from the inner canthi and moved medially. The removed wedge of bone may be either discarded or, if necessary, reinserted to raise the depressed central bony dorsum.

In cartilaginous deformities, isolated sections of septal cartilage are removed submucously. The upper laterals are cut from the septum and the septal cartilage centralized. Sometimes a dorsal cartilage irregularity must be removed submucously and replaced by a piece of straight septal cartilage.

In the dislocated septal cartilage, the mucosa is elevated on one side of the septum and various angles of cartilage are removed. The upper laterals are cut from the septum. The cartilage is elevated from the vomerian floor. The septal cartilage is now centralized and the elevated mucosa replaced. The septal skin is sewn to the columellar skin on each side. Any excess or insufficient upper laterals are now adjusted to the septal cartilage (equalized).

In all these procedures, the perpendicular plate is rarely resected but is usually pushed centrally. Sometimes the vomer is split lengthwise and a section removed before being elevated from the palatal floor. The balance of the vomer is then placed centrally.

Photographs will be shown demonstrating various nasal deviations.

COURSE 410

Room 712 Period Th-1

SAMUEL COHEN, M.D.
Philadelphia, Pa.

Rhinoplasty Complications

Among the postoperative sequelae that will be discussed are the following: flatness of the lower and anterior surfaces of the nasal tip, lower dorsal hump or excessive concavities, postoperative tip drops, intranasal scarring and adhesions, pinched nostrils, retracted columella, unequal nostrils, chronic lymphatic obstruction, and irregular or wide bony nasal dorsums.

COURSE 411

Room 786 Period T-3

JOHN J. CONLEY, M.D.
New York, N. Y.

Modern Concepts in the Treatment of the Pigmented Mole and Malignant Melanoma

This course will emphasize the various types of nevi, their diagnosis, their significance and their treatment. It will also emphasize the care in the diagnosis and treatment of melanoma. Melanoma in the prepubertal and pubertal periods of growth and during pregnancy is discussed in detail. Certain specific etiologic factors and the radical surgical concepts of treatment are outlined. In addition to this, a report will be rendered on cases of melanoma that have been treated with rabies vaccine.

COURSE 412

Room 706 Period T-3 or Th-3

JAMES B. COSTEN, M.D.
St. Louis, Mo.

Mandibular Joint Syndrome: Differential Diagnosis and Treatment

The recognition of a commonly occurring group of pain reflexes due to abnormal function of the mandibular joint is of equal interest to the otolaryngologist, the neurosurgeon and the diagnostician.

The routine examination needed to confirm a diagnosis embraces:

1. Local findings: varieties of malocclusion, loss of teeth, tenderness of mandibular joint to palpation, crepitus within the joints, and trismus

2. X-ray study to show: density changes within the joint, abnormal condylar positions and space relations and erosion of bony landmarks

3. Search for regional conditions which may supply basis for trismus or deranged jaw action

4. Definite relief of pain by fixation of lower jaw with a slight change from its habitual position.

Treatment should be based on:

1. Relief of acute symptoms by baking of the jaws or use of diathermy, mild sedation, and fixation of the lower jaw by elastic, in a position of comfort, for an adequate period each day—one to two hours

2. Recognition of sources of trismus, and removal where possible of impacted teeth, abscessed teeth, parotitis and parotid tumor, infections of jaws, pharynx or neck

3. Completely balanced occlusion

4. Myotonic exercises for an adequate period after restorative dentistry, until unbalanced muscle action is synchronized.

A summary in detail will be presented, using a few selected cases which are typical of the mandibular joint neuralgia.

COURSE 413

Room 706 Period W-1 or Th-2

MAURICE H. COTTLE, M.D.
Chicago, Ill.

Septal Reconstruction Surgery

Deformities of the nose frequently interfere with normal breathing by producing nasal obstruction and changing the course, quantity, and pressure of the air currents. They are caused chiefly by injuries and by developmental abnormalities and occur in any and all parts of the nose, especially in the septum and in the external nasal pyramid. The correction of these deformities should be done by those thoroughly trained in all phases of rhinology.

Reconstruction of the septum rather than resection, together with recognition of associated pathology, has greatly enhanced the possibilities of obtaining satisfactory results. Surgery of one part of the nose should entail a consideration of surgery on other parts of the nose. One cannot correct a hump nose and fail to take into consideration a deviated septum. One cannot do septal surgery and fail to take into consideration the lateral walls of the nose and the relationship of the nasal chambers to the nostrils and the spaces between the upper lateral cartilages and the septum. One cannot disregard the physiology of the turbinates and the anatomy and state of the sinuses, irrespective of what surgical procedure one attempts to perform in the nose. The disturbances of the entire nasal structure must be integrated into one problem. Experience has shown that septal reconstruction is the keystone of adequate treatment.

COURSE 414

Room 711 Period T-1 or W-1

KENNETH L. CRAFT, M.D.
Indianapolis, Ind.

Nasal Allergy: Diagnosis and Medical and Surgical Treatment

I. Brief resume of common symptoms of nasal allergy

II. Diagnostic procedures in nasal allergy
- A. History
  Family. Past and present history of patient — subjective symptoms.
  A thorough history is the most valuable single diagnostic procedure in the examination.
- B. Examination of Nose
  Gross appearance of nasal membranes.
  Contrast with appearance in infection.
- C. Roentgen Examination
  Typical picture.
  Variations.
  Transillumination.
- D. Cytology of Nasal Secretions
  Types of cells found in nasal smear.
  Importance of differentiation.
  Significance of eosinophils.
  Technic.
- E. Hematology
  Interpretation of various cytologic and hematologic tests.
- F. Nasal Polyps
  Always suggest allergy.
  Differentiation between allergic and nonallergic polyps.
- G. Skin Tests
  Patch, scratch, intradermal.
  Choice of allergens to be used in any given case.
  Interpretation and limitation of skin tests.

III. Treatment of nasal allergy
- A. Nonspecific
  1. Local
  2. Systemic
     Internal medication.
     Care of complicating systemic pathology, emotional upsets, etc.
- B. Specific treatment
  1. Prophylaxis
  2. Hyposensitization
     Principles. Indications. Technic.

C. Surgery in nasal allergy.
IV. Case reports demonstrating points discussed in diagnosis and treatment.

COURSE 415

Room 711 Period T-2 or W-2

JAMES E. CROUSHORE, M.D.
Detroit, Mich.

Hemorrhage: Prevention and Control

Heredity and features relating to the chemical and biological properties of blood, both in health and disease, influence bleeding. The various hemostatic agents now available are of value when properly employed. Spontaneous hemorrhage, hemorrhage due to trauma, and hemorrhage during and after surgery will be considered.

Blood supply to the various regions will be illustrated by lantern slides. Ligation in severe hemorrhage will be discussed in detail.

Problems relating to blood transfusion are important. When should blood be given, how much, and how often? How may reactions be avoided and combated when encountered?

COURSE 416

Room 705 Period T-3 or Th-2

STUART C. CULLEN, M.D.
Iowa City, Iowa
BY INVITATION

Anesthesia for Otolaryngology

The course is designed to bring to the attention of those using local anesthetic drugs and depressant drugs, basic and practical factors influencing the safe and satisfactory use of these drugs. The participant in the course may expect to find answers to questions about such things as the proper time to use morphine, how to determine dosage and how to treat overdosage. Other drugs such as barbiturates will be discussed from the standpoint of how they may be used by the practitioner in his office and hospital practice.

Details about local anesthetic drugs including criteria for the evaluation of new preparations, safety factors governing the application of these drugs, and recognition and treatment of reactions will be presented. There will be a discussion of the employment of curare and other muscle relaxants in special situations in otolaryngologic practice. Anesthetic methods and agents for particular problems such as fenestration procedures will be outlined.

COURSE 417

Room 724 Period T-1 or W-1

KENNETH M. DAY, M.D.
Pittsburgh, Pa.

What the Otologist Can Do for Deafness

The object of the course will be to summarize the various conditions causing deafness and to discuss the efficacy and limitations of medical and surgical therapy for its relief. We shall also discuss the value and suitability of hearing aids. There will be some discussion of (a) measures for preventing pathologic ear conditions in infancy, (b) management of secretory otitis, or fluid ears, (c) the effect of radical surgery on hearing, (d) the uses of prostheses, (e) chronic catarrhal otitis, (f) otosclerosis, (g) types of nerve deafness, (h) hearing aids, (i) psychology of deafness and problems of adjustment.

COURSE 418

Room 713 Period W-3 or Th-3

EUGENE L. DERLACKI, M.D.
Chicago, Ill.

Allergy of the Ear

The one-time revolutionary allergic approach to the management of many previously considered surgical problems in rhinology is an accepted fact in our specialty. Now, a similar plan of allergic therapy, aimed not only at treating symptoms but also at the fundamental cause of several acute and chronic disorders of the external, middle and internal ear, is evolving. That allergic diseases do occur in the ear is not a new concept, but general acceptance and recognition by the otologist of the frequency of allergic states in the ear is not as yet an accomplished fact. The diagnosis and treatment of aural allergy is best illustrated by typical case reports.

COURSE 419

Room 728 Period W-3 or Th-3

DAVID D. DeWEESE, M.D.
Portland, Ore.

The Problem of the Dizzy Patient

This course will attempt to cover the evaluation of the patient who comes to the physician's office with the complaint of dizziness. Some time will be spent with the classification from a symptomatic point of view with particular reference to history. The practical value of audiometer and office caloric tests will be mentioned. Vari-

ous accepted treatments which have been proved of value will be discussed. Certain aspects of different types of nystagmus will be covered. The approach to this discussion will be primarily that of the practicing clinician.

COURSE 420

Room 728 Period W-2 or Th-2

JOHN B. ERICH, M.D.
Rochester, Minn.

Fractures of the Facial Bones

This course deals with fractures involving the mandible, maxilla, malar and nasal bones. The methods of treatment which have been found most effective at the Mayo Clinic will be stressed, and classifications which facilitate the management of some of these traumatic injuries will be discussed.

COURSE 421

Room 728 Period T-1 or Th-1

NOAH D. FABRICANT, M.D.
Chicago, Ill.

Modern Medication of the Nose and Sinuses

In an era of uncontrolled usage of antibiotics and antihistaminics there is greater need than ever for employing medication of the nose and sinuses in a sound, rational manner. Effective use of drugs in acute and chronic infections of the nose and sinuses requires knowledge of such supporting clinical disciplines as physiology, pharmacology, histopathology and bacteriology.

Recent advances in the field of local and systemic medication as well as time-tested experiences with the older drugs will be reviewed and evaluated in acute and chronic nasal and sinus disease—the common cold, acute and chronic sinusitis, hypertrophic rhinitis, atrophic rhinitis, nasal allergy, and nasal vestibulitis.

COURSE 422

Room 728 Period T-2 or W-1

J. BROWN FARRIOR, M.D.
and
RICHARD A. BAGBY, M.D.
BY INVITATION
Tampa, Fla.

Surgical Anatomy of the Ear

The purpose of this course is to present the surgical anatomy of the ear in relation to the specific surgical procedures, correlating the x-ray anatomy with the surgical approach. The x-ray anatomy is presented as it is viewed clinically in the four classical roentgen views. The x-ray anatomy in acute mastoiditis is presented. The internal anatomy of the mastoid is discussed in relation to the simple mastoidectomy. The x-ray anatomy of the chronic mastoid is presented and this is correlated with the internal landmarks of the mastoid and the middle ear in relation to the modified radical and radical mastoidectomy. The x-ray anatomy of the temporal bone is discussed in relation to the fenestration operation and the internal anatomy of the mastoid and middle ear are discussed in relation to the fenestration operation. The routes of pneumatization of the petrous apex are presented and the types of petrous apicectomy are discussed in relation to the anatomy of the temporal bone. The anatomy of the facial nerve and facial nerve surgery is presented, the types of labyrinthectomy outlined and the technic of the Day labyrinthotomy is shown.

The course is illustrated by a series of kodachrome slides of x-rays and surgical anatomical dissections.

This course has developed through the Home Study Courses program and is designed to benefit the Board candidates as well as the otologic surgeon.

COURSE 423

Room 710 Period T-3 or Th-1

J. BROWN FARRIOR, M.D.
and
HAROLD CARRON, M.D.
BY INVITATION
Tampa, Fla.

The Fenestration Operation: Anesthetic and Surgical Technic

The purpose of this course is to present the practical points in operating room technic which we think would be of value to other otologic surgeons. Doctor Carron will make a twelve-minute presentation of the anesthetic technics. A few minutes will be allowed for questions. For the remainder of the hour, kodachrome slides will show the various steps in the fenestration operation, giving minute details which reduce the margin of error in fenestration surgery.

This course will describe the surgical team; the crescentic endaural incision; the primary elevation of the flap; the atticoantrostomy approach; the primary and secondary debridement of the flap; the fenestra nov-ovalis with hollow-ground margins; the primary skin graft; and the surgical dressing.

COURSE 424

Room 728 Period T-3
or or
Room 726 Period W-3

GILBERT E. FISHER, M.D.
Birmingham, Ala.

Esophageal Obstruction: Conservative and Surgical Management

This course is a presentation of the conservative and surgical management of the common obstructive lesions of the esophagus. Esophageal manifestations of pellagra, esophageal web, lye burns, achalasia, diverticulum, congenitally short esophagus, carcinoma, benign new growths, and tracheoesophageal fistula are some of the subjects which will be discussed and illustrated with lantern slides.

COURSE 425

Room 724 Period T-2
or or
Room 706 Period W-3

STANTON A. FRIEDBERG, M.D.
Chicago, Ill.
BY INVITATION

Diseases of the Larynx

Various forms of laryngeal pathology will be illustrated by means of kodachrome views. There will be a discussion of the pathogenesis and management of the more common benign lesions such as polyps, vocal nodules, contact ulcers, papillomas and the granulomas which develop after endotracheal anesthesia. The problems involved in leukoplakia will be considered along with some of the diagnostic aspects of laryngeal carcinoma.

COURSE 426

Room 734 Period T-1 or W-1

WILLIAM D. GILL, M.D.
San Antonio, Texas

External Otitis

1. Classification of various clinical types
2. Etiology
3. Otomycosis and its importance in external otitis
4. Classifications of fungi found in otomycosis.
5. Various methods of therapy available at present
6. Fungicides and their degree of effectiveness

COURSE 427

Room 733 Period T-1 or W-1

HARRY N. GLICK, M.D.
St. Louis, Mo.

Surgical Approaches on the Neck

A lecture demonstration on some of the more important operative procedures on the neck, of interest to the otolaryngologist, will be presented. Lantern slides, models, photographs, and carefully prepared dissections will be used to illustrate the various procedures under discussion. An opportunity will be afforded for the inspection and study of the teaching material.

COURSE 428

Room 724 Period T-3 or Th-3

ARAM GLORIG, JR., M.D.
Washington, D. C.

An Evaluation of Methods for Measuring Auditory Acuity

Each method of measurement—past and present—will be presented with a discussion of its uses and relative values.

The discussion will be followed by a demonstration of advanced testing equipment and new equipment developed for special needs.

The purpose of the course is to acquaint the practicing otolaryngologist with practical and advanced methods of testing auditory acuity, as well as to point out some of the limitations of the commonly accepted methods.

COURSE 429

Room 716 Period T-1 or W-1

JOSEPH L. GOLDMAN, M.D.
New York, N. Y.

Rhinologic Diagnosis and Treatment: Newer Criteria

This course will demonstrate the greater value of newer criteria in the diagnosis of nasal and sinus disease in contrast to the older standard methods. These criteria will be especially concerned with diagnostic lavage of sinuses and the bacteriologic and cytologic data obtained from nasal and sinus secretions. It will be shown how such information can be correlated in a practical way and be used clinically to differentiate the infectious from the noninfectious (vasomotor) rhinologic disorders. The application of these data to therapy, particularly the antibiotics, will be stressed.

COURSE 430

Room 726 Period T-3 or Th-3

VICTOR GOODHILL, M.D.
Los Angeles, Calif.

Management of Tinnitus

The patient with tinnitus is frequently a distressing problem to the otologist. The multiple organic causes of tinnitus and the complex psychosomatic patterns encountered make it impossible for any one approach or therapy to alleviate this problem.

A broad psychodynamic approach and certain limited specific therapeutic measures will do much to clarify this confused otologic issue.

A systematic orderly method of analysis is presented as a guide to diagnosis and as a plan for the management of the patient with tinnitus.

COURSE 431

Room 716 Period T-2 or W-2

O. E. HALLBERG, M.D.
Rochester, Minn.

Nosebleed and Its Management

In the treatment of nosebleed, every effort should be made to identify the bleeding point, the principal object being to stop the flow of blood as soon as possible.

A knowledge of the anatomy of the blood supply of the nose is of practical advantage in the decision as to which method to employ in treating the hemorrhage.

Treatment may vary considerably, from simple cautery to ligation of arteries. Supportive treatment with the administration of blood, rutin and vitamins C and K also will be discussed in detail.

Experience reflected in this study was gained in the treatment of 212 patients at the Mayo Clinic.

COURSE 432

Room 716 Period T-3 or Th-1

JAMES A. HARRILL, M.D.
Winston-Salem, N.C.

Bronchography

The indications, contraindications, technic and complications of bronchography in adults and children will be discussed. A technic as used in adults, showing positioning of patient and x-ray views taken, will be demonstrated by motion pictures. The various pathologic lesions demonstrated by bronchography will be discussed by illustrative cases.

COURSE 433

Room 731 Period T-3
or or
Room 734 Period W-2

JEROME A. HILGER, M.D.
St. Paul, Minn.

Therapeutic Approaches to the Inner Ear

Two decades ago salient contributions to the pathology of inner ear deafness were made. Within the past decade the pathology of advanced hydrolabyrinth was added. It was attributed to a single disease entity (Meniere's disease, endolymphatic hydrops or other synonyms).

As medical knowledge of neurovascular change and endocrine imbalance increases it becomes increasingly clear that inner ear vertigo and deafness do not have a single genesis; that the labyrinthine endpathology, even that of hydrops, does not denote a single etiology; and that therapy, useful only in the formative stage, must vary to meet the cause. A stereotyped therapeutic approach must inevitably be futile in a disorder of multiple etiologies.

The causes and treatment of some of these inner ear malfunctions are discussed.

COURSE 434

Room 736 Period Th-2

JEROME A. HILGER, M.D.
St. Paul, Minn.

Direct Fixation of Facial Fractures

Facial fractures are frequently extremely complex and varied. In almost all instances, however, they are reducible to relatively simple terms. Their realignment and fixation can be accomplished by a variety of satisfactory and acceptable methods. As a basic principle to the solution of the individual problem, simplicity of the method should be the basic theme. Frequently, this simplicity is best obtained by direct wire fixation of the component fragments. To accent and elaborate some of the simple, direct approaches is the purpose of this hour.

COURSE 435

Room 715 Period W-2

FREDERICK T. HILL, M.D.
Waterville, Maine

Antro-Alveolar Fistulas

While many antro-alveolar fistulas may respond to conservative measures, at times the management of this condition may tax

the ingenuity of the specialist. Control of any infection of the maxillary antrum is essential and, in early cases, may suffice, allowing spontaneous closure of the fistula. In cases of longer duration some plastic procedure usually will be required. The type of operation should be determined by the conditions present in each case. The technic of the reversed flap plastic, applicable to large defects, will be demonstrated.

COURSE 436

Room 715 Period Th-2

FREDERICK T. HILL, M.D.
Waterville, Maine

Essentials for Aural Rehabilitation

Rehabilitation of the deafened should be a responsibility of the otologist. While the ideal program is an elaborate one involving extensive personnel and finances, the need for such a program may deprive many handicapped individuals of their opportunity to become self-supporting citizens. As rehabilitation is most effective in the early childhood years, postponing the inauguration of such a program until something approaching the ideal is possible means that much valuable time is irrevocably lost. A simple program, doing with what is possible, thinking in terms of service rather than facilities, may be productive of much good. Such a program, "on a shoestring," will be described.

COURSE 437

Room 716 Period W-3

WILLIAM J. HITSCHLER, M.D.
Philadelphia, Pa.

Cardiac Arrest and Resuscitation from the Viewpoint of the Otolaryngologist

Cardiac arrest is occurring or being recognized more often than formerly. It may occur in relatively minor operations (tonsillectomy), as well as in major ones. Resuscitative procedures such as cardiac massage have proved successful in many cases.

This course concerns itself with the early recognition of cardiac arrest, its causes and prevention, and the procedures necessary for the restoration of the circulation and of the normal heart beat. It is presented from the viewpoint of a physician, such as the otolaryngologist, who is not trained in thoracic surgery. A colored motion picture (on the dog) is included.

COURSE 438

Room 715 Period T-3 or W-3

PAUL H. HOLINGER, M.D.
Chicago, Ill.

Pediatric Laryngology

In this course the management of diseases of the larynx in infants and children will be presented. Beginning with methods of examination, the important technics of x-ray visualization will be discussed with particular attention directed toward the roentgen interpretation of etiology and site of respiratory obstruction. The problems of direct laryngoscopy will be considered, with a discussion of diagnostic and therapeutic technics. A resume of congenital and acquired lesions of the larynx in infants and children will be shown by means of slides and color motion pictures.

COURSE 439

Room 715 Period T-1 or Th-3

PAUL H. HOLINGER, M.D.
Chicago, Ill.

Esophageal Diseases in Infants and Children

Esophageal diseases in infants and children fall into three main categories—those due to congenital anomalies, to the ingestion of caustics, and to foreign bodies. These conditions and other less frequently seen lesions will be discussed with particular stress placed upon the esophagoscopic phases of both diagnosis and treatment. Instrumentarium used in esophagoscopic procedures in infants will be shown. The discussion of the various disease entities will be illustrated by slides and color motion pictures.

COURSE 440

Room 727 Period W-1 or Th-1

EDGAR M. HOLMES, M.D.
Boston, Mass.

Scars of the Face: Correction and Repair

Before undertaking the correction of scars of the face, it is necessary to fully understand the fundamentals of wound healing and how disfiguring scars arise. The principles of wound healing will therefore be considered, mentioning the reaction of tissues to injury and the normal processes which take place in wound repair. With this for a basis, the various conditions which affect wound healing will be discussed and evaluated.

Many disfiguring scars could have been prevented had certain fundamentals been followed. These fundamentals will be dealt with. Questions such as: When should a wound be closed? How much cleaning should be done? Should skin be discarded to create a straight line? What sutures should be employed? will be answered. The various types of scars that are encountered, that is, the wide, keloid, contracted and depressed scars, will be considered. Mention will be made of the various causes of undesirable scars so that these pitfalls may be avoided as much as possible. Specific procedures will then be discussed for the correction of disfiguring scars. The proper method of excising scars, the importance of being sure that tension is relieved, the proper use of Z incisions and local flaps will be dealt with.

COURSE 441

Room 740 — Period T-1 or W-1

ROBERT M. HOSLER, M.D.
Cleveland, Ohio
BY INVITATION

The Emergency Treatment of Cardiac Arrest in the Operating Room

Cardiac arrest in the operating room is the enigma and challenge of present day surgery and anesthesia. Obviously the best therapy for this catastrophe is in its prevention; however, to be successful in meeting this challenge when suddenly confronted with it, one must know reflexly the things to do and the things not to do. (One is just as important as the other.) One does not have time to improvise methods, as a time limitation of three to five minutes must be overcome.

Practical, precise, and detailed methods will first be presented concerning the reestablishment of the oxygen system. Secondly, the methods of restoration of the heart beat in cardiac standstill and ventricular fibrillation will be presented. These are the two conditions which are met in the operating room.

A colored motion picture will demonstrate these procedures and the Rand-Beck defibrillating machine will be on exhibition.

COURSE 442

Room 722 — Period T-1
or
Room 744 — Period Th-1

HOWARD P. HOUSE, M.D.
Los Angeles, Calif.

Surgery of the Chronically Discharging Ear

The indications for surgical intervention and the operation of choice in the chronically discharging ear will be presented.

A detailed description of the surgical technic used in both the modified and the radical mastoid operation will be given. The necessity of a definite detailed pattern of surgical approach in these cases will be emphasized. Many points of practical value in mastoid surgery, which will aid in preventing undue surgical complications, will be presented. The postoperative management of these cases will be reviewed.

This course in chronic ear surgery will be fully illustrated by slides and kodachrome motion pictures.

COURSE 443

Room 740 — Period T-3 or W-3

ROBERT E. HOWARD, M.D.
Cincinnati, Ohio

Practical Suggestions for Office Procedures in the Ear, Nose, and Throat

1. Management of the patient
2. Arrangement of treatment rooms
3. Proper sterilization of instruments and supplies
4. Special examination and treatments
   a. Routine ear, nose and throat examinations
   b. Antrum irrigations
   c. Tubal catheterization
   d. Minor surgical procedures in the office
   e. Type of anesthesia used for above procedures
   f. Allergy—skin tests and treatment
   g. X-ray for diagnosis
5. Bookkeeping for comparative studies

COURSE 444

Room 715 — Period T-2
or
Room 782 — Period W-2

LELAND G. HUNNICUTT, M.D.
Pasadena, Calif.

The Chronically Congested Nose

Various causes for the chronically congested nose will be discussed. A regimen for nasal hygiene will be outlined, and treatment for an airway obstructed by a polypoid inferior turbinate will be presented. Relief for stasis of secretions will also be considered.

COURSE 445

Room 781 Period T-1 or Th-1

LELAND G. HUNNICUTT, M.D.
Pasadena, Calif.

When and How to Make Tissue Examination: Practical Office Concept

Emphasis is placed on knowing the normal tissue, both macroscopic and microscopic. There are normal variations of the tissues, and to the trained eye and mind changes beyond this are recognized disease. At this point tissue examination must be made. Methods of tissue examination are by biopsy, scrapings, Papinicolaou stain and smears and culture. These observations and methods of study of tissue as done in the office will be presented.

COURSE 446

Room 727 Period Th-2

LESTER T. JONES, M.D.
Portland, Ore.

Tear Sac Surgery: The Intranasal Approach

As a basis for this study, a series of one hundred consecutive intranasal dacryocystorhinostomies will be used. This technic and its advantages will be demonstrated by the use of motion pictures, slides, and anatomic specimens.

Special emphasis will be given to the handling of failures resulting from previous tear sac surgery where this or any other method has been used. This will include a simplified technic for inserting polyethylene tubing in the lacrimal passages to maintain patency.

COURSE 447

Room 781 Period W-2 or Th-2

RAYMOND E. JORDAN, M.D.
Pittsburgh, Pa.

Management of Otologic Conditions Complicated by Allergy

An analysis of 727 consecutive otologic patients revealed that 27 per cent had otologic conditions caused or complicated by allergy. From this survey it is apparent that allergy has a definite role in the practice of otology. Allergy was found in 85 per cent of the patients with chronic secretory otitis media. Persistent discharge in simple types of chronic suppurative otitis media and postoperative modified or radical mastoidectomies can be caused by an allergy. Certain types of tinnitus will respond to allergic management. Some cases of Meniere's disease will respond to minimal subcutaneous doses of histamine and therefore can be considered in the allergic category. The above conditions will be discussed from the standpoint of signs, symptoms, local treatment and allergic management.

COURSE 448

Room 732 Period T-3 or W-3

ARTHUR L. JUERS, M.D.
Miami, Fla.

Hearing Tests

The use and abuse of masking are discussed. The significance of the contour of pure tone air conduction audiometric curves is illustrated by selected case records. Particular emphasis is placed on the correlation of tuning fork tests, pure tone air-bone threshold relationship and speech tests in the selection of patients as to suitability for fenestration.

COURSE 449

Room 781 Period T-3 or W-3

JACQUELINE KEASTER
Iowa City, Iowa
BY INVITATION

Speech and Hearing in Children Under Five Years of Age

Discussion in this course will include the following points:

1. The normal development of speech in young children, including the babbling period, the development of words and sentences, with time devoted to standards of articulation considered to be within the normal range
2. Speech retardation and its possible causes
3. Diagnostic procedures, including tests of hearing appropriate for the evaluation of hearing acuity in young children
4. Indications for training, including the use of hearing aids with borderline as well as severe hearing losses

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COURSE 450

Room 781 Period W-1 or Th-3

GEORGE KELEMEN, M.D.
Boston, Mass.

Mechanical Injuries of the Hearing Organ

A survey will be given on mechanical aural injury on the basis of pathology. The

material is exclusively of human origin. Dealing with fractures of the skull involving the temporal bone, preference will be given to the presentation of emergencies as they occur in ordinary life, caused by a blow, by a fall from an elevation, by fast moving vehicles. Stress will be laid on the role of mechanical birth trauma with far-reaching effects in the postnatal period. Proceeding through external, middle and internal ear, the pertaining methods of diagnosis and treatment will be discussed step by step.

COURSE 451

Room 776 Period T-1 or W-1

JOSEPH I. KEMLER, M.D.
Baltimore, Md.

Mirror Laryngology

Examination of the larynx in the indirect method will be taken up in detail. Advantages as well as shortcomings will be discussed. Operative work consisting of removal of material for biopsy, benign growths, and the use of cautery in the office for some lesions will be demonstrated by motion picture and x-ray films.

The purpose of this presentation is, first, to familiarize the younger man with the thorough examination of the larynx in the office and, second, to show the possibility and the feasibility of taking biopsies by the indirect method in the office so that there will be no delay in diagnosing malignancy.

COURSE 452

Room 786 Period W-2
or or
Room 736 Period Th-3

CHARLES E. KINNEY, M.D.
Cleveland, Ohio

Acoustic Trauma

The presently known facts and experimental work on this subject will be summarized and presented in a practical fashion. Methods of control of noise production and methods of defending the ear against noise trauma will be discussed.

Audiometric tests for the determination of the extent of hearing handicaps and one's susceptibility to noise injury will be described.

COURSE 453

Room 776 Period T-2 or Th-2

JOHN A. KIRCHNER, M.D.
New Haven, Conn.

Difficulty in Swallowing

The significance of this complaint will be evaluated. Differential diagnosis and treatment of conditions with dysphagia as a presenting symptom will be discussed. Neurologic syndromes of which dysphagia is a component will be reviewed. The normal swallowing mechanism in the normal subject will be illustrated by lantern slides and motion picture studies of barium swallow-fluoroscopy. The abnormalities of deglutition will likewise be shown in motion picture fluoroscopic studies of neurologic lesions involving the oral and pharyngeal musculature; myasthenia gravis; tumors of the pharynx and cervical esophagus; cardiospasm; pharyngeal diverticula; congenital abnormalities; hysterical and functional dysphagia.

COURSE 454

Room 776 Period W-3 or Th-3

CLAIR M. KOS, M.D.
Iowa City, Iowa

Clinical Orientation in the Management of Dizziness

"Dizziness" is due to such a multiplicity of disturbances that the presentation of the symptom excites a variety of perplexing diagnostic considerations.

Some of these variations, however, do not receive the distinctive recognition they deserve both for prognostic and therapeutic assessment simply because certain significant clinical features may easily escape detection. Excluding those pathologic states which can be accounted for by otoscopic evidence of disease or by rather obvious neurologic signs, a group of labyrinthine disturbances occur to challenge even the most resolute efforts to explain their nature. In those instances in which the "dizziness" does not indicate or suggest serious consequences to the patient's longevity and for which no exact cause may be readily ascribed, the compliment of symptoms comprised of vertigo, tinnitus, hearing loss, and occasionally nystagmus, and the associated autonomic phenomena, is a familiar clinical disorder generally classified as Meniere's symptom complex.

The functional aberrations thus classified often trace a symptomatic pattern upon which one may place relatively high diagnostic and prognostic value. Detailed history, physical examination and the functional tests constitute informational sources of varying importance in each case.

Such findings often resolve into discrete categories suggesting a more acceptable operational classification for diagnosis, prognosis and treatment than the term "Meniere's symptom complex" ordinarily implies. It becomes apparent after extensive clinical study correlated with available laboratory data that most of these disturbances are different manifestations attributable to vasomotor irregularities or cytochemical alterations involving both oxygen and nutritional transport in the functional segments of the temporal bone. Since such disturbances do not lend themselves to complete investigative accessibility either on the operating table or in the laboratory, it is illuminating to refer to available supporting evidence provided by investigations in this and some of the related fields of study.

Such studies suggest three principal clinical classifications of so-called "idiopathic dizziness"; labyrinthine hydrops, labyrinthine ischemia, and labyrinthine apoplexy. These, of course, may necessarily be differentiated from many other conditions such as hypostatic syncope, multiple sclerosis, eighth nerve tumors, aneurysms, vasospasm, and occlusion of cerebellar and brainstem vessels which may mimic, if only temporarily, the group of labyrinthine disturbances.

COURSE 455

Room 776 Period W-2 or Th-1

HUGH A. KUHN, M.D.
Hammond, Ind.

Allergic Diseases of the Ear

The problem of allergies as they relate to and disturb the comfort of the ear will be discussed from several angles. Allergic hearing defects are easy to observe, diagnose and treat, and a quick response follows accurate management. An appreciable number of patients with lowered hearing due to allergies has been found. The frequent combination of pathology and allergy represents a better opportunity for a hearing restoration than management only of the infectious process. The symptoms of aural allergies are dependent on the points of the hearing mechanism involved.

COURSE 456

Room 786 Period W-1 or Th-1

FRANCIS E. LeJeune, M.D.
New Orleans, La.

The Technic of Intralaryngeal Surgery

This is a continuation of the course given several years ago on Suspension Laryngoscopy.

The first phase to be dealt with for successful intralaryngeal surgery is that of anesthesia; the type as well as the technic will be discussed in detail.

Consideration will be made of benign growths, including hemangiomas, polyps, papillomas, cysts and simple edema or hydrops of the cords, accompanied by a motion picture showing the technic.

A discussion will be held on leukoplakia and its removal, early malignancy of the cords and the procedure of removal which has given the best results.

Finally the procedure of intralaryngeal arytenoidectomy will be considered.

COURSE 457

Room 740 Period W-2 or Th-2

MAURICE LENZ, M.D.
New York, N. Y.
BY INVITATION

Indications for Radiotherapy in Cancer of the Nose and Throat

X-rays and radium rays damage cancer and normal tissues. Radiotherapy can be successful if the cancer is more radiosensitive than the irradiated normal tissue. Cancer growing in muscle, cartilage, bone or fat is difficult to arrest by radiotherapy. Cancerocidal effect of radiotherapy is limited to cancer cells exposed to adequate radiation dosage. Correct clinical localization of cancer is easiest, and radiotherapy is most successful in accessible superficially growing cancer of limited extent without tendency to local invasion or regional metastasis. Infection reduces tolerance of cancerocidal radiation and should be combated before initiating radiotherapy. Chondroradionecrosis is serious except in sites such as the epiglottis where the radiation slough is easily disposed of.

The results of radiotherapy in cancer of the nose and throat are best in lymphosarcoma, lymphoepithelioma, and certain epitheliomas; they are worse in adrenocarcinoma, and surgery should usually be preferred to radiotherapy in fibrosarcomas, myosarcomas, chondrosarcomas, osteosarcomas, or melanosarcomas.

The above principles of radiotherapy of cancer are illustrated by clinical experience with this treatment at the Manhattan Eye, Ear and Throat and Presbyterian Hospitals, New York.

COURSE 458

Room 727 Periods T-3 or Th-3

MICHAEL L. LEWIN, M.D.
New York, N. Y.

Supporting Grafts in Facial and Nasal Reconstructive Surgery: Evaluation and Technic

In reconstructive surgery around the face, particularly the nose, there are frequent indications for restoration of contoural defects. These are not only of a skeletal nature but may be due to losses of soft tissue, as, for example, in depressed scars of the cheek. A great variety of connective tissue transplants have been used and are recommended in the literature to the confusion of the reader. Alloplastic material of an inorganic nature, such as tantalum, or plastics such as polyethylene have many strong advocates.

The insertion of a transplant may be all that is required for correction of a depressed nasal deformity, but often small supporting grafts to the dorsum or columella are part of a rhinoplastic procedure. Likewise, the correction of a receding chin may be performed independently or in combination with a rhinoplasty for improvement of the facial profile. Supporting grafts are also called for in the correction of depressions in the frontal or maxillo-malar region.

The purpose of this course is to examine the biological behavior of different transplantation materials and discuss their surgical applications. Autogenous rib and septal cartilage, cancellous bone, derma and other transplantation material will be considered. Surgical technics will be discussed both in respect to procurement and utilization of the grafts. The course will be illustrated by lantern slides demonstrating cases and procedures.

COURSE 459

Room 744 Period T-3 or W-3

PERRIN H. LONG, M.D.
Brooklyn, N. Y.
BY INVITATION

The Use of Chemotherapeutic and Antibiotic Agents in Treatment of Diseases of the Ear, Nose and Throat

The multiplicity of chemotherapeutic and antibiotic agents which are available today for the treatment of infections of the nose and throat, and of complications arising from such infections, makes it imperative to select the most effective agent if treatment is to be in the best interests of the patient. It is also highly important to evaluate the possibilities for the use of these agents alone in the treatment of otolaryngologic infections and also in combination with competent surgical procedures. The rational use of these compounds requires a working knowledge of their clinical pharmacology, toxicology, and range of action when used alone or in combination.

In the course of this exercise, the points which have been outlined above will be discussed at length, and adequate time will be provided for answering questions.

COURSE 460

Room 710 Period T-1 or W-1

JAMES H. MAXWELL, M.D.
Ann Arbor, Mich.

Tumors of the Face and Neck in Infancy and Childhood

Minor irregularities in the embryonic growth pattern produce anomalies which rather frequently are manifested in infants and children as soft tissue tumors and cysts of the face and neck. It is with such teratologic lesions that this discussion is concerned primarily.

Grouped according to their anlagen, lesions derived from (1) the vascular system, (2) the nervous system, (3) the branchial structures, (4) the thyroglossal duct, and (5) faults in midline fusion will be discussed in regard to their differential diagnosis and treatment. Lantern slides illustrating the various lesions will be shown.

COURSE 461

Room 732 Period W-1 or Th-1

FREEMAN McCONNELL, Ph.D.
Nashville, Tenn.
BY INVITATION

Speech Audiometry for the Otologist

The value of speech audiometry as a diagnostic tool for otologists has been increasingly demonstrated in recent years. The usefulness of a single quantitative measure of a person's practical ability to hear speech and the relationship between the speech reception threshold and the pure

tone audiogram will be discussed. Discrepancy between pure tone and speech thresholds can be significant diagnostically. Testing of speech discrimination is also an extremely useful adjunct to the diagnosis and evaluation of hearing problems. Effective use of simplified speech audiometric equipment in the otologist's offices will be described.

COURSE 462

Room 750 Period T-1 or W-1

JAMES W. McLAURIN, M.D.
Baton Rouge, La.

Principles of Therapy in Otitis Externa

External otitis is a perennial problem of the otolaryngologist, particularly in the South and Southwest. It is not a problem that has been solved, but we have come a long way toward its solution. Believing no one medicinal agent ever has been or ever will be satisfactory for all infections of the external auditory canal, the general principles that should be followed in handling such infections will be covered. The remaining time will be spent on questions regarding specific treatment.

COURSE 463

Room 777 Period W-1 or Th-1

W. J. McNALLY, M.D.
Montreal, Que., Canada

Practical Methods of Carrying Out Labyrinthine Tests: Indications and Clinical Applications

This course is designed to present the basic or elementary facts which are necessary for an understanding of clinical labyrinthine tests. Much of the time will be taken up in studying the actual methods of carrying out the various tests. Time will also be given to working out the interpretation of reactions which follow the simple tests. The simplicity of the tests will be stressed because they can be made very complex. There is still so much speculation and so much to be learned about labyrinthine function that there is no universal agreement about many of the points which come up in the course of a labyrinthine examination. There are, however, a sufficient number of basic facts known to justify the general adoption of a labyrinthine test which should become a part of the otologist's routine examination. As a brief introduction, the anatomy and physiology of the semicircular canals and the otoliths will be reviewed. The examination of spontaneous phenomena will be considered. Guides for the detection of spontaneous nystagmus will be given. An interpretation of past-pointing tests and their relation to vestibular stimulation will be studied. The technic of performing the caloric and rotation tests will be taken up in detail. The rules which govern labyrinthine reactions will be used to interpret the results of these tests. Finally, the significance of the results of the tests in labyrinthine and intracranial diagnosis will be investigated.

COURSE 464

Room 777 Period T-2 or Th-2

ALDEN H. MILLER, M.D.
Los Angeles, Calif.

Laryngeal Emergencies in Infants and Children

This course will be a practical presentation of the diagnosis and treatment of conditions causing dangerous obstruction of the larynx and trachea in infants and children.

The acute obstructive infectious diseases of the larynx, supraglottic or subglottic, cause most emergencies, and a brief differential diagnosis and outline of treatment will be given.

A number of less common conditions are frequently presented to the otolaryngologist for immediate diagnosis and treatment. Of these the following will be discussed.

A. Newborn and infants
   1. Laryngomalacia (congenital laryngeal stridor)
   2. Glossoptosis and micronathia
   3. Tumors, trauma, paralysis
   4. Dyspnea due to tracheal compression by mediastinal tumors or anomalies as distinguished from atelectasis, bronchiolitis, agenesis, etc.

B. Children
   1. Bulbar poliomyelitis
   2. Head injuries
   3. Fracture of the larynx
   4. Foreign bodies and caustic burns
   5. Angioneurotic edema

COURSE 465

Room 777 Period W-3 or Th-3

PAUL M. MOORE, JR., M.D.
Cleveland, Ohio

Diseases of the Maxillary Sinus: Medical and Surgical Treatment

1. The maxillary sinus will be considered

from the anatomic and physiologic aspects.

2. The management of acute and chronic infections will be considered from both the medical and surgical standpoints.
3. The various surgical procedures which have proved to be of value will be discussed in detail.

COURSE 466

Room 777 Period T-3 or W-2

LEWIS F. MORRISON, M.D.
San Francisco, Calif.

The Technic of Tonsillectomy

The technic of tonsillectomy under general anesthesia is not presented under the guise of something revolutionary or new. It is a selection of the better parts of many methods combined to produce a routine that is simple, direct, and almost foolproof. Lantern slides and a short kodachrome motion picture illustrate the advantages of maximum visibility and a dry field.

COURSE 467

Room 713 Period W-1 or Th-2

HARRY NEIVERT, M.D.
New York, N. Y.

The Care of Instruments

A craftsman should know his tools. The cleansing, sharpening and care of instruments should be of interest to all surgeons. Instruments that are sent away to be sharpened are returned only after a long delay. Many are ground away unnecessarily, resulting in early replacement. Knowing how to sharpen instruments with simple cheap tools saves money and tempers and makes work easier.

Those taking the course should bring with them curets, scissors and knives they want sharpened. (They should be able to save enough to pay the dues of the Academy.) They will also learn how to make instruments for daily use from simple materials.

COURSE 468

Room 750 Period T-2 or W-2

JOSEPH H. OGURA, M.D.
St. Louis, Mo.

Laryngectomy and Neck Dissection

The technic of the one-stage widefield laryngectomy combined with a radical neck dissection in one block will be detailed on serial lantern slides to demonstrate the most efficient way for the removal of the primary malignancy and its metastasis.

The importance of the sequence of steps in the procedure, conservation of bleeding, and preservation of as much function as possible will be emphasized.

The basis of the selection of cases for surgery will be discussed. Case illustrations will be given.

COURSE 469

Room 750 Period T-3 or Th-3

PETER N. PASTORE, M.D.
Richmond, Va.

A Lump in the Throat

Patients often present themselves to the otolaryngologist with such symptoms as a lump in the throat, scratchy or itchy throat, recurrent sore throat and persistent nonproductive cough.

Palpation of the cervical structures and hurried inspection of the pharynx frequently fail to explain such symptoms.

The sensory nerve supply of the pharynx and the mechanism thought to be responsible for such symptoms will be discussed and supported with case reports and illustrations.

COURSE 470

Room 713 Period T-2 or W-2

SAMUEL J. PEARLMAN, M.D.
Chicago, Ill.

The Tonsil and Adenoid Operation

Preoperative Considerations
1. Indications for surgery
2. Time of year
3. Poliomyelitis influence
4. Severity of status

Operative Considerations
1. Type of anesthesia
2. Advantages and disadvantages of various technical procedures
3. Control of bleeding
4. Complications
   a. Respiratory arrest
   b. Aspiration of clots
   c. Excessive bleeding
   d. Influence of weather

Postoperative Considerations
1. Immediate postoperative care
2. Early bleeding
   a. Tonsil
   b. Adenoids
3. Medication
   a. Pain
   b. Hemorrhage
4. Diet

5. Delayed bleeding:
   a. Causes
   b. Therapy
6. Late Complications
   Adhesions
   Recurrence

COURSE 471

Room 784 Period T-2 or Th-1

HENRY B. PERLMAN, M.D.
Chicago, Ill.

Serous Otitis

Fluid in the middle ear may be found with a number of different clinical conditions. Examples of these various clinical states will be presented together with kodachrome slides of the associated tympanic membrane changes. Drum changes associated with complete filling of the middle ear will be demonstrated. Experiences with serous otitis media in a group of children will be analyzed. The role of the eustachian tube in this disease will be discussed and motion pictures of the tube in action will be shown.

COURSE 472

Room 784 Period W-2 or Th-2

MAX EDWARD POHLMAN, M.D.
Los Angeles, Calif.

The Plastic Ear Drum

Demonstration of a plastic ear drum, its use as an aid to middle ear deafness; the technic in fitting the prosthesis and the clinical results obtained so far in this type of work.

COURSE 473

Room 782 Period T-3 or Th-3

JOEL J. PRESSMAN, M.D.
Beverly Hills, Calif.

The Rhinologist and the Problem of Rhinoplasty

The course is intended to help the rhinologist decide whether or not he shall do rhinoplasty. It will outline a logical routine of steps to follow in developing skill, types of cases to select in order safely to gain experience, the complications one will encounter and practical points of value of aid in establishing oneself as a rhinoplastic surgeon.

Nothing of a technical nature will be discussed. The course is designed for those not yet doing this type of surgery but who are considering the advisability of doing it.

COURSE 474

Room 782 or Room 705 Period T-2 or Period Th-3

F. JOHNSON PUTNEY, M.D.
Philadelphia, Pa.

Surgery of Neck Tumors

The practical aspects of surgery of benign and malignant neoplasms of the neck, excluding the thyroid and larynx, are presented. Epidermoid tumors of the neck are diagnostic problems and it is always necessary to search elsewhere about the head and neck for primary lesions before definitive surgical treatment of the neck lesion. The surgery of metastatic cervical nodes is discussed in detail with emphasis on the technic of radical neck dissection.

COURSE 475

Room 782 Period T-1 or W-3

SCOTT N. REGER, Ph.D.
Iowa City, Iowa

Detection of Auditory Malingering in Office Practice

This course will consider (1) the legal status of the expert witness; (2) relationships between the ear history, physical examinations, vestibular and hearing tests; and (3) demonstration of certain specific unilateral and bilateral hearing tests.

COURSE 476

Room 782 or Room 752 Period W-1 or Period Th-1

SCOTT N. REGER, Ph.D.
Iowa City, Iowa

Practical Office Audiometry

This course will consider (1) the common sources of error in audiometry; (2) an adequate testing procedure; (3) the use of masking while obtaining air and bone conduction measurements; and (4) interpretation of audiograms.

COURSE 477

Room 784 Period T-1 or W-1

LYMAN G. RICHARDS, M.D.
Brookline, Mass.

Helpful Suggestions Concerning Office Practice

1. Reception of the patient
   a. Waiting room arrangements
   b. Receptionist technic

c. Secretarial contact
d. Arrangement of consulting room
e. Treatment rooms
f. Cashier contact

2. Treatment room fixtures, chair, cabinets, compressed air suction
3. Methods of anesthesia
4. Technics of specific examinations
   a. Larynx examination
   b. Nasopharyngeal examination: transillumination
   c. Antrum irrigations
   d. Audiometric setup
   e. Tubal catheterization
   f. Minor operative procedures such as nasal polypectomy, myringotomy, turbinal cauterization
   g. Physiotherapy with diathermy
   h. Remainder of time for discussion of common therapeutic problems, such as otitis externa and postnasal catarrh
   i. Role of psychosomatic approach in otolaryngology

COURSE 478

Room 784 Period W-3 or Th-3

GILBERT J. ROBERTS, M.D.
Pomona, Calif.

The Management of Nasal Fractures

Nasal fractures all too frequently are dealt with hurriedly and inadequately, leaving an unsatisfactory cosmetic and functional result.

A clear understanding of the anatomy of the external nose, particularly the relationship between the bony nasal arch and the cartilaginous structure, is essential to the successful management of nasal fractures.

A technic permitting deliberate manipulation under adequate local anesthesia, followed by adequate splinting, inside and out, will be presented, accompanied by lantern slides and demonstration of instruments and splints.

COURSE 479

Room 715 Period W-1 or Th-1

SAM E. ROBERTS, M.D.
Kansas City, Mo.

Painful Head and Neck Syndrome

It is my observation that approximately 90 per cent of the severe recurring chronic headaches are of vascular origin either from vasodilatation or vasospasm. The latter has not been emphasized nearly enough. These patients come to us because they assume that their headaches are caused by sinusitis. When one realizes that the vascular system is controlled through the autonomic nervous system with the hypothalamus as the control station, it becomes evident that dysfunctions in this region are the chief etiologic background. It is well to use the term "dysfunctions" because we do not know exactly what produces hypofunction or hyperfunction of these nerves and their controlling center. The hypothalamus controls practically all body rhythm. It is easy to visualize either a vasodilatation or a vasospasm on the basis of a dysfunctioning of the rhythm.

Since the patient consults us for these disturbances, it behooves us to prepare ourselves to advise him intelligently and to treat him efficiently.

COURSE 480

Room 740 Period T-2 or Th-1

ROBERT E. RYAN, M.D.
St. Louis, Mo.

Headache

The physiologic basis of head pain will be discussed. Following this the history-taking of the headache patient is gone over along with the routine examination usually necessary for the average headache patient. The objectives of all headache treatment are then pointed out. The differential diagnosis and treatment of various types of headache will then be presented. Among those discussed will be migraine, histaminic cephalalgia, sinusitis, myalgia, psychogenic headache, tension headache, hypertensive headache, generalized vasodilating headache, otic, ocular and the mixed type of headache.

COURSE 481

Room 713 Period T-3 or Th-1

JOSEPH SATALOFF, M.D.
Philadelphia, Pa.

Present Treatment of Nerve Deafness

1. Optimistic approach to the problem
2. Psychology of hearing loss
3. Aural rehabilitation
4. Instruction in making ear impressions (Several impressions will be made in the classroom.)
5. Usefulness and limitation of hearing aids
6. If time permits, special problems in dealing with the child with nerve deafness

COURSE 482

Room 726 Period T-1
or or
Room 740 Period Th-3

ALBERT P. SELTZER, M.D.
Philadelphia, Pa.

New Concepts and Technics in Plastic Surgery of the Nose and Face-Lift Operation

The lecturer will discuss the complete technic in nasoplastic surgery, including basic principles, demonstrated by clear illustrations, starting from the selection of instruments, anesthetic, and step-by-step procedure to the postsurgical result. He will talk on the anatomy of the nose together with the technic in the use of new instruments (original). Esthetic nasal surgery will be discussed in detail. Elimination of fixity of facial expressions, grafts and their preparation, the unsuccessful operation, its causes and prevention, will also be discussed. A kodachrome film will present several patients before and after surgery in which the newer instruments and technics were employed. Errors in plastic surgery of the nose will be discussed in detail.

Time permitting, complete details of the face-lift operation will be given, with particular emphasis on facial paralysis and operation for esthetic purposes. Presurgical preparation, indications and contraindications, one-stage versus two-stage operations, anesthesia, various types of incisions, rotation and fixation of flaps, complications, dressings, correction for upper and lower eyelid pouches, correction for forehead-glabellar and nasal-labial wrinkles, and correction of sagging jowls will all be discussed in detail and demonstrated by slides.

COURSE 483

Room 721 Period W-1 or Th-1

BEN H. SENTURIA, M.D.
St. Louis, Mo.

Treatment of External Otitis

Much uncertainty exists as to the etiology and pathology of external otitis. As a consequence, there are many therapeutic agents generally employed in the treatment of external otitis which have little scientific basis for their use.

The application of controlled bacteriologic and mycologic procedures has demonstrated that our concepts of etiology need revision. Preliminary histopathologic observations have produced further data of importance in evaluating the treatment of infections of the external auditory canal.

On the basis of these findings a practical clinical classification of external otitis has been set up. The therapeutic agents and procedures currently in use for the treatment of the various categories of external otitis will be examined and discussed.

COURSE 484

Room 721 Period T-3 or Th-3

GEORGE E. SHAMBAUGH, JR., M.D.
Chicago, Ill.

Failures in Fenestration Surgery

A failure is defined as an operation which fails to obtain the anticipated reduction in the air-bone gap. The chief causes for failure will be described with the relative proportion of each, in a follow-up study of 3000 consecutive operations. Methods of preventing failures will be described with special emphasis on the selection of cases for operation, the prevention of osteogenic closure, and the prevention of postoperative serous labyrinthitis.

COURSE 485

Room 736 Period T-1 or Th-1

JOHN J. SHEA, M.D.
Memphis, Tenn.

Intranasal Surgery

The art of intranasal surgery is fading with the passing of the generation of otolaryngologists who were trained forty years ago. An attempt will be made to describe the pathology and the various intranasal operations which were commonly used but which now are so seldom demanded that many of the younger otolaryngologists have missed the opportunity of observing them.

COURSE 486

Room 705 Period W-3 or Th-1

BENJAMIN H. SHUSTER, M.D.
Philadelphia, Pa.

Diagnosis and Management of Ocular Conditions of Otologic and Nasal Origin

There are a number of patients who come to the otolaryngologist for evaluation of eye conditions which may have been caused by an ear or nasal infection. Lacrimation, orbital infections and swellings, and sudden onset of blindness are examples. These conditions will be discussed. There are also

some borderline symptoms like headache, vertigo, nystagmus, paralysis of ocular muscles and visual field disturbances which require an otolaryngologic evaluation and management. These, too, will be classified and discussed.

COURSE 487

Room 726 Period T-2

MARVIN W. SIMMONS, M.D.
Fresno, Calif.

Intranasal Dacryocystorhinostomy

Surgery of the tear sac belongs in the field of the nasal surgeon but is neglected by too many rhinologists. The operation is much less complicated than its name. In this course emphasis will be placed upon a simple, very safe and successful technic which will give great personal satisfaction to the operator. Any specialist familiar with the routine of nasal surgery will find little difficulty learning the procedure.

COURSE 488

Room 732 Period W-2
or or
Room 733 Period Th-1

KINSEY M. SIMONTON, M.D.
Rochester, Minn.

Recruitment of Loudness and Diplacusis Binauralis

The course will consist of a discussion of recruitment of loudness and diplacusis binauralis as well as the other signs which indicate deafness due to dysfunction of the cochlear end-organ. The mechanism of the phenomena will be outlined as a background for the clinical applications. Tests for these phenomena are of value in the differential diagnosis of endolymphatic hydrops from lesions of the trunk of the acoustic nerve, and in determining the phase of activity of endolymphatic hydrops. Knowledge of the phase of activity of endolymphatic hydrops aids in determining the prognosis of hearing and the result of treatment. The course will be illustrated by data obtained from patients; methods of testing for recruitment and diplacusis will be demonstrated.

COURSE 489

Room 781 Period T-2
or or
Room 795 Period Th-2

J. DUDLEY SINGLETON, M.D.
Dallas, Texas

The Tonsil and Adenoid Problem in Children

In the management of the tonsil and adenoid problem in children, the physician should assume that normal healthy tonsils and adenoids serve some useful purpose. Their removal should be advised only after careful study convinces one that they are chronically diseased, and that their continued presence is causing or may cause sufficient injury to the health of the child to justify the dangers encountered in their removal.

The subject of tonsils and adenoids in children will be discussed under the following topics:

1. Selection and preparation of patients for operation
2. Choice of anesthesia
3. Technic of operation
4. Postoperative care of patients
5. Prevention and control of primary and secondary hemorrhage
6. Tonsillo-adenoidectomy in the allergic child
7. Tonsillectomy and poliomyelitis
8. Tonsillo-adenoidectomy and impaired hearing, case reports with audiograms before and after operation

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COURSE 490

Room 784 Period T-3
or or
Room 789 Period W-2

WILLIAM C. THORNELL, M.D.
Cincinnati, Ohio

Intralaryngeal Arytenoidectomy in Bilateral Vocal Cord Paralysis

Various external operations have been described for the correction of bilateral abductor vocal cord paralysis. This course will deal with the intralaryngeal approach for arytenoidectomy which has been successfully applied in cases of bilateral abductor vocal cord paralysis. The surgery is carried out through the Lynch laryngeal suspension apparatus under combined local and intravenous pentothal sodium with curare.

The various modifications that have been introduced as well as complications to be considered will be discussed. Lantern slides will be used to demonstrate the technic of operation, and recordings of the voice taken before and after surgery will be presented.

COURSE 491

Room 721 Period T-1 or W-3

G. EDWARD TREMBLE, M.D.
Montreal, Que., Canada

Facts and Fallacies of Nose and Thoat Medication

In the last twenty years, with our better understanding of nasal physiology and pathology, there has been a trend towards conservatism in the treatment of infections of the nose and throat.

Many of the commonly used medications today are, however, outdated as they fail to satisfy basic requirements, while others are grossly overrated.

The restoration of the physiologic changes produced in the disease process and the recognition of the limitations of the various medications used are of primary importance.

Throat medications have always been very popular among laymen but their value is questionable.

These factors will be thoroughly discussed. The lecture will be followed by a short colored motion picture on ciliary action.

COURSE 492

Room 750 Period W-3 or Th-1

KURT TSCHIASSNY, M.D.
Cincinnati, Ohio

BY INVITATION

A Method of Locating the Lesion in Facial Nerve Paralysis

Factors of fundamental importance in differentiating between upper and lower motor neuron lesions will be discussed. Six different syndromes depending on the simultaneous presence or absence of taste in the chorda tympani area, lacrimation, and stapedius reflex will be described and analyzed with regard to their individual significance for lesions located at six different levels in the lower motor neuron. A few illustrative lantern slides will be shown.

COURSE 493

Room 795 Period T-2 or W-2

O. E. VAN ALYEA, M.D.
Chicago, Ill.

Embryology of the Ear, Nose, and Throat

I. Embryonic Stage

During the first eight weeks of intrauterine life, the structures which compose the ear, nose, and throat are in the formative stage of development. The major changes which take place at this time will be considered step by step by means of lantern slides and wax reconstructions.

II. Fetal Stage

The changes which occur from the eighth week are of especial interest to the otolaryngologist. In this course considerable time will be devoted to the study of serial sections made from fetal heads of various ages with special emphasis on those periods which deal with the origin and development of the nasal sinuses.

III. Congenital Defects

These occur largely as a result of growth defects in the early formative stages of the embryo. Anomalies of the various types encountered in clinical practice will be studied by means of lantern slide projections of charts, x-rays, photographs and specimens from the cadaver.

COURSE 494

Room 795 Period T-3 or Th-3

O. E. VAN ALYEA, M.D.
Chicago, Ill.

Modern Management of Chronic Sinusitis

Persistence of a sinus infection implies inadequate drainage. Factors contributing to drainage impairment may be local or systemic. The local factors may be anatomic or pathologic in nature and in some cases the sinusitis is maintained by a combination of contributing causes.

The goal of the rhinologist is to eliminate the causative factors while treating the infection. Most cases respond to simple measures, such as infraction of the middle turbinate, or the removal of a few polyps along with the control of allergy. Progress in treatment of the cases is noted by occasional roentgenologic studies with iodized oil in the sinus and by examination of the exudate removed by irrigation. In those cases which fail to respond readily to carefully directed treatment there is usually some overlooked contributing systemic or local factor. In certain cases thickened mucosa lines the sinus ostium to such an extent as to occlude, or nearly occlude, the drainage outlet and extra drainage openings (windows) may be required.

Seldom is the radical removal of sinus mucosa in the treatment of sinusitis indicated, and it should be instituted with the knowledge that only temporary relief may be expected and that a return of the symptoms, even more pronounced than formerly, may result.

In this course the technics of various therapeutic measures used successfully in the management of sinus disease will be discussed in detail.

COURSE 495

Room 795 Period W-1 or Th-1

HANS VON LEDEN, M.D.
Chicago, Ill.

Tracheotomy: Its Present Concept

Indications for tracheotomy will be discussed, including its recent use in bulbar poliomyelitis, tetanus, crushing chest injuries, anesthetic and postoperative complications, cranial and spinal injuries, and lower respiratory diseases. Tracheotomy in preventing fatal aspiration in comatose or debilitated patients will be given consideration.

The technic of the procedure will be illustrated, with special reference to the avoidance of complications. Differences in surgical technic in children and adults will be mentioned. The problem of an emergency tracheotomy will be treated separately. The postoperative care of tracheotomized patients will be outlined and stressed.

COURSE 496

Room 721 Period T-2 or Th-2

THEODORE E. WALSH, M.D.
St. Louis, Mo.

Acute and Chronic Otitis Media: Treatment

1. The etiology of acute otitis media will be discussed.
2. The pathogenesis and pathologic changes in acute otitis media will be reviewed with special attention to the healing processes in the mastoid air cells.
3. The rational treatment in each stage of the disease will be suggested.
4. A classification of chronic otitis media in relation to the dangers of the condition will be offered.
5. Therapy, "medical" and "surgical," will be discussed.

COURSE 497

Room 795 Period T-1 or W-3

W. W. WILKERSON, JR., M.D.
Nashville, Tenn.

A New Approach to a Hearing and Speech Center For Your City

Surveys reveal that at least 5 per cent of our school children have hearing difficulties and that 11 per cent have speech deficiencies. Are you or is your community prepared to cope with this problem?

The formation of a Hearing and Speech Center will better the lives of those individuals handicapped with hearing and speech defects, will improve public relations, will increase the prestige of the otologists, and will aid in making your city a medical center.

Objectives of a Hearing and Speech Center:

1. Examination of preschool children, school children and adults for hearing and speech defects
2. Medical and surgical care as needed
3. Administration of hearing and speech training
4. The fitting of hearing aids and training in their use
5. The training of teachers for teaching handicapped children in grade school
6. Establishment of courses for graduate students in this specialty

All of these objectives obviously could apply to only a few centers. Sponsorship and financial support of a Hearing and Speech Center will be discussed in detail.

COURSE 498

Room 710 Period W-3 or Th-3

LAWRENCE R. BOIES, M.D.
Minneapolis, Minn.

JEROME A. HILGER, M.D.
St. Paul, Minn.

THEODORE RASMUSSEN, M.D.
Chicago, Ill.
BY INVITATION

Head Pain: Round Table Discussion

The task of differential diagnosis of the etiology of head pain as a chronic problem is a very common experience to the otolaryngologist.

In the past, the etiology of chronic head pain was often attributed to some form of disease within the nasal space when an obvious cause was lacking. Modern observations have changed that concept.

A classification of the causes of headache and neuralgia will be presented. Attention will be focused on those which arise in the nasal space. Differential diagnosis and treatment will be stressed. Questions will be encouraged.

COURSE 499

Room 710 Period W-2 or Th-2

LOUIS H. CLERF, M.D.
F. JOHNSON PUTNEY, M.D.
Philadelphia, Pa.

DANIEL C. BAKER, M.D.
New York, N. Y.

Diseases of the Larynx, Neck, Tracheobronchial Tree and Esophagus: Round Table Discussion

This period will be devoted to an informal discussion, on a question and answer basis, of diseases involving the larynx, neck, tracheobronchial tree and esophagus.

If you have any problems, real or imaginary, that occur in your everyday practice in the fields of laryngology or bronchoesophagology, bring them with you for presentation to the Problem Panel which will try to help you. Provisions will be made for you to submit written questions during the panel discussions.

COURSE 500

Room 736 Period W-1
or or
Room 744 Period Th-3

A. C. FURSTENBERG, M.D.
Ann Arbor, Mich.

GORDON B. NEW, M.D.
Rochester, Minn.

LeROY A. SCHALL, M.D.
Boston, Mass.

Tumors and Infections of the Neck: Round Table Discussion

COURSE 501

Room 710 Period T-2
or or
Room 786 Period W-3

PHILIP E. MELTZER, M.D.
FRANK D. LATHROP, M.D.
M. H. LURIE, M.D.
and
FRANCIS WEILLE, M.D.
BY INVITATION
Boston, Mass.

Otological Problems: Round Table Discussion

The hour will be given over for an unprepared discussion with the men taking the course. They are free to participate in a stimulating and informal exchange of views concerning practical everyday problems commonly met with in our practice. The panel will be prepared to discuss all types of treatment and surgery of the ear. The registrants are urged to contribute to the discussion.

COURSE 502

Room 731 Period W-3 or Th-3

ROBERT E. PRIEST, M.D.
Minneapolis, Minn.

NOAH D. FABRICANT, M.D.
Chicago, Ill.

HAROLD WRIGHT, Ph.D.
Minneapolis, Minn.
BY INVITATION

Medications for the Otolaryngologist: Round Table Discussion

The purpose of this course is to allow physicians attending the Academy to discuss questions concerning drugs with these well-known authorities. It is believed that this "Pharmacology Forum" will provide an interesting and profitable hour for those who elect to bring their drug problems to it.

COURSE 503

Room 738 Period W-1 or Th-1

O. E. VAN ALYEA, M.D.
Chicago, Ill.

ANDERSON HILDING, M.D.
Tenafly, N. J.

J. M. ROBISON, M.D.
Houston, Texas

Rhinology: Round Table Discussion

During the past twenty-five years considerable research and fine thinking has dispelled much of the confusion which has always been associated with nasal sinuses.

That problems still exist is evidenced by the fact that whenever sinuses come up for discussion there are always differences of opinion. This panel will be an open forum and a free expression of opinions from the floor will be encouraged. A panel board will guide the discussion and attempt to answer questions directed to it.

The Following Are Condensed Schedules of Courses Previously Described

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Allen, J. H., et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
2	Ascher	Aqueous Veins in Glaucoma	2 PERIODS W-5, W-6
3	Bedell	Medical Ophthalmoscopy	4 PERIODS M-5, T-5, W-5, Th-5
4	Berliner	Slit Lamp Microscopy—Advanced	4 PERIODS M-4, T-4, W-4, Th-4
5	Birge, et al	New Theories — Retinal Venous Thrombosis	2 PERIODS Th-4, Th-5
6 or 7	Boeder	Aniseikonia & Space Eikonometry	2 PERIODS W-4, W-5 or Th-4, Th-5
8	Brown, et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
9	Clark	Surgery to Ocular Muscles	2 PERIODS M-5, M-6
10 or 11	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
12	Costenbader, et al	Concomitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
13	DeVoe	Clinical Application of Gonioscopy	2 PERIODS Th-5, Th-6
14	Elwyn	Heredodegenerative Diseases of Retina	2 PERIODS M-5, M-6
15	Fink	Management of Oblique Muscle Problems	3 PERIODS T-5, W-5, Th-5
16	Hardy, et al	Basic Principles of Orthoptics	4 PERIODS M-4, T-4, W-4, Th-4
17	Hastings	Psychosomatic Medicine	2 PERIODS W-5, W-6
18 or 19	Henderson	Perimetry	2 PERIODS M-4, M-5 or Th-4, Th-5
20	Hughes, et al	Plastic Surgery	10 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, Th-4, Th-5
21	Jardon	Fitting of Ocular Prostheses	2 PERIODS W-5, W-6
22 or 23	Johnson, L. V.	Aqueous Tonography	2 PERIODS W-5, W-6 or Th-4, Th-5
24 or 25	Johnson, V. C.	Diagnostic Value of X-ray	2 PERIODS M-5, M-6 or T-5, T-6
26	Knighton, et al	Highlights of Glaucoma	8 PERIODS M-5, M-6, T-5, T-6, W-5, W-6, Th-5, Th-6
[illegible]	Kuhn	Industrial Ophthalmology	3 PERIODS M-4, T-4, W-4

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
28	Lebensohn, et al	Refraction	6 PERIODS W-4, W-5, W-6, Th-4, Th-5, Th-6
29	Lowrey	Military Ophthalmology	2 PERIODS W-5, W-6
30	Lyle	Neuro-Ophthalmology	4 PERIODS M-4, T-4, W-4, Th-4
31	McLean, et al	Cataract Surgery	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
32	Meek	Surgical Anatomy of Orbit	2 PERIODS T-5, T-6
33	Meyer, et al	Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
34	Ogle	Essential Aspects of Binocular Vision	2 PERIODS M-5, M-6
35	O'Rourke	Manifest & Cycloplegic Refraction	2 PERIODS T-5, T-6
36	Paton	Corneal Surgery	3 PERIODS T-4, W-4, Th-4
37	B. Payne, et al	Practical Eye Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
38	Pino	Bifocal Lenses & Presbyopia	2 PERIODS M-5, M-6
39	Romaine, et al	Ocular Motility	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
40 or 41	Schepens	Ophthalmoscopy in Diagnosis of Retinal Detachment	2 PERIODS M-5, M-6 or T-5, T-6
42	Scobee, et al	Eye Muscles	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
43	Stow, et al	Applied Clinical Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
44	Theobald, et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
45	Thorpe, et al	Slit Lamp Biomicroscopy	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
46	Vail & Minsky	Zonular Membrane & Cataract Surgery	2 PERIODS M-5, M-6
47	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-5, M-6, T-5, T-6, W-5, W-6
48	Wiener	Practical Points in Surgery	3 PERIODS M-6, T-6, W-6
49	Friedenwald	Measurement of Aqueous Flow	2 PERIODS W-4, W-5

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
204	Atkinson	Measures in Cataract Extraction
205	Baird	Acute Lesions of Cornea
206	Baker	Neuralgias of Head
212	Byrnes	Adjuncts in Teaching Ophthalmology
219	Cordes	Surgery of Congenital Cataracts
226	Falls	Ocular Geriatrics
235	Gipner	Recent Advances
245	Hogan	Pathology of the Choroid
256	Lloyd	Macular Lesions
262	McCaslin	Ocular Anesthesia
268	Osher	Diagnostic Approach to Squint Surgery
269	Owens & Owens	Retrolental Fibroplasia
272	Pfeiffer	Diagnosis with Aid of X-ray
274	Post, L. T.	Difficult Problems in Refraction
275	Post, M. H.	Operating Room Technic
280	Rychener	Lesions of the Lids
285	Spaeth	Lacrimal Gland & Sac
297	Whalman	Minor Surgical Procedures of Eye
A	Home Study	Embryology, Anatomy & Neuro-Anatomy

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
207	Barkan	Congenital Glaucoma
209	Berens	Uveitis: Etiology & Treatment
220	Cusick	Ptosis
224	Dunnington	Ocular Wound Healing
229	Fread	Ideal Refraction Equipment
252	Leinfelder	Papilledema & Optic Neuritis
255	Linksz	Visual Physiology
266	Newell	Rabbit Surgery
278	Rudolph	Advantages of General Anesthesia
290	Swan	Value of New Drugs
294	Veasey	Refraction Difficulties
B	Home Study	Physiology

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
201	Alvaro	Uveitis: Etiology & Treatment
203	Amenabar	Defachment of Retina: Treatment
216	Chace	Ocular Malingering
222	Davies	Clinical Pathology in Optic Atrophy
223	Dunlap	Divergence Anomalies
231	Fry	Differential Diagnosis of Lesions at Posterior Pole of Eye
233	Gemeroy	External Diseases of Eye
239	Gray	Intraocular Foreign Bodies
243	Hanser	Ocular Allergy
253	Lemoine	Vitreous Opacities
254	Lepard	Eye Diseases Common to Children
261	Marshall	Lid Infections
270	Payne, F.	Unilateral Visual Failure
277	Reese	Cataract Extraction
279	Rundles	Streak Retinoscopy
281	Scheie	Surgery in Congenital Glaucoma
C	Home Study	Optics & Principles of Biomicroscopy

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvis	End Results of Cataract Wound Closure
204	Atkinson	Measures in Cataract Extraction
205	Baird	Acute Lesions of Cornea
206	Baker	Neuralgias of Head
217	Chandler	Secondary Glaucoma in Aphakia
228	Fralick	External Ocular Manifestations of General Disease
235	Gipner	Recent Advances
240	Guibor	Physiology of Ocular Muscles
256	Lloyd	Macular Lesions
257	Lueck	Practical Optics
262	McCaslin	Ocular Anesthesia
269	Owens & Owens	Retrolental Fibroplasia
272	Pfeiffer	Diagnosis with Aid of X-ray
275	Post, M. H.	Operating Room Technic
283	Siniscal	Chronic Lid Conditions
286	Spaeth	Plastic Surgery
297	Whalman	Minor Surgical Procedures of Eye
D	Home Study	Refraction

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
209	Berens	Uveitis: Etiology & Treatment
210	Berke	Surgery of Oblique Muscles
220	Cusick	Ptosis
224	Dunnington	Ocular Wound Healing
227	Falls	Practical Application of Heredity
229	Fread	Ideal Refraction Equipment
252	Leinfelder	Papilledema & Optic Neuritis
255	Linksz	Visual Physiology
276	Randolph	Handling Patients with Advanced Glaucoma
282	Simpson	Convergence Insufficiency & Divergence Excess: Office Management
291	Tait	Methods in Correction of Accommodation & Convergence Abnormalities
294	Veasey	Refraction Difficulties
296	Walsh	Interesting Neuro-Ophthalmologic Cases
E	Home Study	External Diseases, Bacteriology & Virology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
201	Alvaro	Uveitis: Etiology & Treatment
203	Amenabar	Detachment of Retina: Treatment
216	Chace	Ocular Malingering
222	Davies	Clinical Pathology in Optic Atrophy
223	Dunlap	Divergence Anomalies
231	Fry	Differential Diagnosis of Lesions at Posterior Pole of Eye
233	Gemeroy	External Diseases of Eye
239	Gray	Intraocular Foreign Bodies
243	Hanser	Ocular Allergy
246	Iliff	Beta Irradiation Therapy
253	Lemoine	Vitreous Opacities
261	Marshall	Lid Infections
270	Payne, F.	Unilateral Visual Failure
273	Pischel	Retinal Detachment Surgery: Complications
279	Rundles	Streak Retinoscopy
281	Scheie	Surgery in Congenital Glaucoma
287	Steffensen	ACTH & Cortisone
F	Home Study	Perimetry & Neuro-Ophthalmology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvis	End Results of Cataract Wound Closure
211	Bettman	Value of Goniotomies
213	Calhoun	Dystrophies & Degenerations of Cornea
217	Chandler	Secondary Glaucoma in Aphakia
221	Cutler	Procedures Used in Enucleation
238	Gordon	Anatomy of Orbital Fascia
241	Guibor	Neuromotor Defects of Ocular Muscles
244	Harrington	Psychosomatic Phases
247	Jobe	Practical Physiologic Optics
263	McGuire	Heterophoria & Heterotropia
271	Perera	Retinal Detachment
283	Siniscal	Chronic Lid Conditions
284	Smith	Practical Points in Office Treatment
292	Theodore	Ocular Therapeutics
298	Wiener	Minor Lid Surgery
G	Home Study	Extraocular Muscles

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Changes in Crystalline Lens
210	Berke	Surgery of Oblique Muscles
215	Cassady	Dacryocystitis of Infancy
232	Gailey	Complications of Cataract Surgery
234	Gifford	Dacryocystorhinostomy
237	Goar	Dystrophies of Cornea
249	Klien	Clinical Pathology of Retinal Lesions
264	Morrison	Office Management of Glaucoma
267	Olson	Iritis, Iridocyclitis, & Keratitis
291	Tait	Methods in Correction of Accommodation & Convergence Abnormalities
293	Trueman	Cerebral Lesions
H	Home Study	Ophthalmoscopy & Medical Ophthalmology

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
214	Callahan	Repair of Lid Lacerations
218	Cholden	Informing Patient of Blindness: Psychiatric Aspects
225	Egan	Cross Cylinder Application & Theory
236	Givner	Systemic Disease
246	Iliff	Beta Irradiation Therapy
248	Katz	Corneal Dystrophies
250	Krewson	Clinical Significance of Oblique Muscles
251	Kronfeld	End Results in Glaucoma Surgery
254	Lepard	Eye Diseases Common to Children
258	Macnie	Head Trauma with Ocular Significance
259	Mangold	Proper Fitting of Glasses
260	Mann, M.	Hypertensive Changes in Fundus
265	Mullen	Ophthalmic Emergencies
287	Steffensen	ACTH & Cortisone
288	Stimson	Lenses for the Aphakic
299	Mann, M.	Refraction and Lens for Cataract Patient
I	Home Study	Pathology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
211	Bettman	Value of Goniotomies
213	Calhoun	Dystrophies & Degenerations of Cornea
221	Cutler	Procedures Used in Enucleation
238	Gordon	Anatomy of Orbital Fascia
242	Guibor	Neurology of Ocular Muscles
244	Harrington	Psychosomatic Phases
247	Jobe	Practical Physiologic Optics
263	McGuire	Heterophoria & Heterotropia
267	Olson	Iritis, Iridocyclitis, & Keratitis
271	Perera	Retinal Detachment
284	Smith	Practical Points in Office Treatment
292	Theodore	Ocular Therapeutics
J	Home Study	Pharmacology & Therapeutics

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Changes in Crystalline Lens
215	Cassady	Dacryocystitis of Infancy
232	Gailey	Complications of Cataract Surgery
234	Gifford	Dacryocystorhinostomy
249	Klien	Clinical Pathology of Retinal Lesions
258	Macnie	Head Trauma with Ocular Significance
264	Morrison	Office Management of Glaucoma
293	Trueman	Cerebral Lesions

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
214	Callahan	Repair of Lid Lacerations
225	Egan	Cross Cylinder Application & Theory
236	Givner	Systemic Disease
248	Katz	Corneal Dystrophies
250	Krewson	Clinical Significance of Oblique Muscles
259	Mangold	Proper Fitting of Glasses
260	Mann, M.	Hypertensive Changes in Fundus
265	Mullen	Ophthalmic Emergencies
287	Steffensen	ACTH & Cortisone
288	Stimson	Lenses for the Aphakic
299	Mann, W.	Refraction and Lens for Cataract Patients

CONTINUOUS COURSES — OTOLARYNGOLOGY

COURSE	SPEAKERS	TITLE	PERIODS USED
101	Altmann & Waltner	Histopathology of Ear—Part I	2 PERIODS T-1, T-2
102	Altmann & Waltner	Histopathology of Ear—Part II	2 PERIODS W-1, W-2
103 or 104	Bryan & Bryan	Cytologic Pathology of ENT Diseases	2 PERIODS T-1, T-2 or W-1, W-2
105 or 106	Conley	Cancer of Head & Neck	2 PERIODS T-1, T-2 or Th-2, Th-3
107	Converse	Nasal Plastic Surgery (Basic)	2 PERIODS W-1, W-2
108	Converse	Nasal Plastic Surgery (Advanced)	2 PERIODS Th-2, Th-3
109 or 110	Figi	Lesions of Mouth	2 PERIODS T-2, T-3 or W-2, W-3
111	Fowler & Montreuil	Histology of Ear — Vertical Sections	2 PERIODS T-1, T-2
112	Fowler & Montreuil	Histology of Ear — Horizontal Sections	2 PERIODS Th-1, Th-2
113 or 114	Hansel	Allergy	2 PERIODS T-1, T-2 or Th-1, Th-2
115	Jackson	Technic in Peroral Endoscopy	3 PERIODS T-3, W-3, Th-2
116	Kazanjian, et al	Traumatic Injuries of Face	2 PERIODS T-1, T-2
117 or 118 or 119	Lempert	Evaluating Degree of Hearing Function	2 PERIODS T-2, T-3 or W-2, W-3 or Th-2, Th-3
120 or 121	Lindsay	Vertigo	2 PERIODS W-2, W-3 or Th-2, Th-3
122 or 123	Maliniac	Maxillofacial Plastic Surgery	2 PERIODS T-1, T-2 or W-2, W-3
124	Marcus, et al	Hearing Problems in Children	2 PERIODS W-1, W-2
125	Moses	Speech & Voice Therapy	2 PERIODS W-2, W-3
126 or 127	Mueller	Practical Audiometry	2 PERIODS W-2, W-3 or Th-1, Th-2
128 or 129	Orton	Applied Surgery of Neck	2 PERIODS T-2, T-3 or Th-2, Th-3
130 or 131	Sellers	Equilibration	2 PERIODS T-1, T-2 or Th-2, Th-3
132 or 133	Semenov	Therapy in Pathology of ENT	2 PERIODS T-2, T-3 or Th-2, Th-3
134	Tucker, et al	Esophageal Diseases & Foreign Bodies	3 PERIODS T-1, W-3, Th-1
135	Tucker, et al	Laryngeal Surgery	3 PERIODS T-3, W-2, Th-2
136 or 137	Turchik	Nasal Obstruction	2 PERIODS W-1, W-2 or Th-2, Th-3

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Adenoid Operation: Relation to Auditory Apparatus
403	Baron	Modified Radical Mastoidectomy
406	Boyden	Chronic Frontal Sinus Surgery
414	Craft	Nasal Allergy
417	Day	What the Otologist Can Do for Deafness
421	Fabricant	Modern Medication of Nose & Sinuses
426	Gill	External Otitis
427	Glick	Surgical Approaches on Neck
429	Goldman	Rhinologic Diagnosis & Treatment
439	Holinger	Esophageal Diseases in Infants & Children
441	Hosler	Cardiac Arrest During Operation
442	House	Surgery of Chronically Discharging Ear
445	Hunnicutt	When & How to Make Tissue Examination
451	Kemler	Mirror Laryngology
460	Maxwell	Tumors of Face & Neck in Infancy & Childhood
462	McLaurin	Otitis Externa: Therapy
475	Reger	Auditory Malingering
477	Richards	Suggestions Concerning Office Practice
482	Seltzer	Plastic Surgery of Nose & Face-Lift Operation
485	Shea	Intranasal Surgery
491	Tremble	Nose & Throat Medication
497	Wilkerson	Hearing & Speech Center for Your City

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
402	Ashley	Dust Therapy in Respiratory Allergy
404	Becker	Plastic Surgery of Protruding Ears
407	Canfield	Audiology for Practicing Otologist
415	Croushore	Hemorrhage: Prevention & Control
422	Farrior & Bagby	Surgical Anatomy of Ear
425	Friedberg	Diseases of Larynx
431	Hallberg	Nosebleed & Its Management
444	Hunnicutt	Chronically Congested Nose
453	Kirchner	Difficulty in Swallowing
464	Miller	Laryngeal Emergencies in Infants & Children
468	Ogura	Laryngectomy & Neck Dissection
470	Pearlman	Tonsil & Adenoid Operation
471	Perlman	Serous Otitis
474	Putney	Surgery of Neck Tumors
480	Ryan	Headache
487	Simmons	Intranasal Dacryocystorhinostomy
489	Singleton	Tonsil & Adenoid Problem in Children
493	Van Alyea	Embryology of ENT
496	Walsh	Otitis Media: Treatment
501	Panel	Otological Problems

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
405	Bellucci	Submucous Resection of Nasal Septum
409	Cohen	Deviated Noses & Their Correction
411	Conley	Pigmented Mole & Malignant Melanoma: Treatment
412	Costen	Mandibular Joint Syndrome
416	Cullen	Anesthesia
423	Farrior & Carron	Fenestration Operation
424	Fisher	Esophageal Obstruction
428	Glorig	Evaluation of Methods for Measuring Auditory Acuity
430	Goodhill	Management of Tinnitus
432	Harrill	Bronchography
433	Hilger	Therapeutic Approaches to Inner Ear
438	Holinger	Pediatric Laryngology
443	Howard	Office Procedures in ENT
448	Juers	Hearing Tests
449	Keaster	Speech & Hearing in Children Under Five
458	Lewin	Facial & Nasal Reconstructive Surgery
459	Long	Chemotherapeutic & Antibiotic Agents
466	Morrison	Technic of Tonsillectomy
469	Pastore	A Lump in the Throat
473	Pressman	Rhinologist & Problem of Rhinoplasty
481	Sataloff	Treatment of Nerve Deafness
484	Shambaugh	Failures in Fenestration Surgery
490	Thornell	Intralaryngeal Arytenoidectomy
494	Van Alyea	Chronic Sinusitis

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Adenoid Operation: Relation to Auditory Apparatus
403	Baron	Modified Radical Mastoidectomy
408	Carhart	Audiogram in Otologic Diagnosis
413	Cottle	Septal Reconstruction Surgery
414	Craft	Nasal Allergy
417	Day	What the Otologist Can Do for Deafness
422	Farrior & Bagby	Surgical Anatomy of Ear
426	Gill	External Otitis
427	Glick	Surgical Approaches on Neck
429	Goldman	Rhinologic Diagnosis & Treatment
440	Holmes	Scars of Face
441	Hosler	Cardiac Arrest During Operation
450	Kelemen	Mechanical Injuries of Hearing Organ
451	Kemler	Mirror Laryngology
456	LeJeune	Technic of Intralaryngeal Surgery
460	Maxwell	Tumors of Face & Neck in Infancy & Childhood
461	McConnell	Speech Audiometry for Otologist
462	McLaurin	Otitis Externa: Therapy
463	McNally	Labyrinthine Tests
467	Neivert	Care of Instruments
476	Reger	Practical Office Audiometry
477	Richards	Suggestions Concerning Office Practice
479	Roberts, S. E	Head & Neck Syndrome
483	Senturia	External Otitis
495	Von Leden	Tracheotomy
500	Panel	Tumors & Infections of Neck
503	Panel	Rhinology

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
404	Becker	Plastic Surgery of Protruding Ears
407	Canfield	Audiology for Practicing Otologist
415	Croushore	Hemorrhage: Prevention & Control
420	Erich	Fractures of Facial Bones
431	Hallberg	Nosebleed & Its Management
433	Hilger	Therapeutic Approaches to Inner Ear
435	Hill	Antro-Alveolar Fistulas
444	Hunnicutt	Chronically Congested Nose
447	Jordan	Otologic Conditions Complicated by Allergy
452	Kinney	Acoustic Trauma
455	Kuhn	Allergic Diseases of Ear
457	Lenz	Cancer of Nose & Throat: Radiotherapy
466	Morrison	Technic of Tonsillectomy
468	Ogura	Laryngectomy & Neck Dissection
470	Pearlman	Tonsil & Adenoid Operation
472	Pohlman	Plastic Ear Drum
488	Simonton	Recruitment of Loudness & Diplacusis Binauralis
490	Thornell	Intralaryngeal Arytenoidectomy
493	Van Alyea	Embryology of ENT
499	Panel	Diseases of Larynx, Neck, Tracheobronchial Tree & Esophagus

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
405	Bellucci	Submucous Resection of Nasal Septum
409	Cohen	Deviated Noses & Their Correction
418	Derlacki	Allergy of Ear
419	DeWeese	The Dizzy Patient
424	Fisher	Esophageal Obstruction
425	Friedberg	Diseases of Larynx
437	Hitschler	Cardiac Arrest & Resuscitation
438	Holinger	Pediatric Laryngology
443	Howard	Office Procedures in ENT
448	Juers	Hearing Tests
449	Keaster	Speech & Hearing in Children Under Five
454	Kos	Clinical Orientation in Management of Dizziness
459	Long	Chemotherapeutic & Antibiotic Agents
465	Moore	Diseases of Maxillary Sinus
475	Reger	Auditory Malingering
478	Roberts, G. J.	Management of Nasal Fractures
486	Shuster	Ocular Conditions of Otologic & Nasal Origin
491	Tremble	Nose & Throat Medication
492	Tschiassny	Locating Lesion in Facial Nerve Paralysis
497	Wilkerson	Hearing & Speech Center for Your City
498	Panel	Head Pain
501	Panel	Otological Problems
502	Panel	Medications

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
406	Boyden	Chronic Frontal Sinus Surgery
408	Carhart	Audiogram in Otologic Diagnosis
410	Cohen	Rhinoplasty Complications
421	Fabricant	Modern Medication of Nose & Sinuses
423	Farrior & Carron	Fenestration Operation
432	Harrill	Bronchography
440	Holmes	Scars of Face
442	House	Surgery of Chronically Discharging Ear
445	Hunnicutt	When & How to Make Tissue Examination
455	Kuhn	Allergic Diseases of Ear
456	LeJeune	Technic of Intralaryngeal Surgery
461	McConnell	Speech Audiometry for Otologist
463	McNally	Labyrinthine Tests
471	Perlman	Serous Otitis
476	Reger	Practical Office Audiometry
479	Roberts, S. E.	Head & Neck Syndrome
480	Ryan	Headache
481	Sataloff	Treatment of Nerve Deafness
483	Senturia	External Otitis
485	Shea	Intranasal Surgery
486	Shuster	Ocular Conditions of Otologic & Nasal Origin
488	Simonton	Recruitment of Loudness & Diplacusis Binauralis
492	Tschiassny	Locating Lesion in Facial Nerve Paralysis
495	Von Leden	Tracheotomy
503	Panel	Rhinology

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
402	Ashley	Dust Therapy in Respiratory Allergy
413	Cottle	Septal Reconstruction Surgery
416	Cullen	Anesthesia
420	Erich	Fractures of Facial Bones
434	Hilger	Facial Fractures
436	Hill	Aural Rehabilitation
446	Jones	Tear Sac Surgery
447	Jordan	Otologic Conditions Complicated by Allergy
453	Kirchner	Difficulty in Swallowing
457	Lenz	Cancer of Nose & Throat: Radiotherapy
464	Miller	Laryngeal Emergencies in Infants & Children
467	Neivert	Care of Instruments
472	Pohlman	Plastic Ear Drum
489	Singleton	Tonsil & Adenoid Problem in Children
496	Walsh	Otitis Media: Treatment
499	Panel	Diseases of Larynx, Neck, Tracheobronchial Tree & Esophagus

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
412	Costen	Mandibular Joint Syndrome
418	Derlacki	Allergy of Ear
419	DeWeese	The Dizzy Patient
428	Glorig	Evaluation of Methods for Measuring Auditory Acuity
430	Goodhill	Management of Tinnitus
439	Holinger	Esophageal Diseases in Infants & Children
450	Kelemen	Mechanical Injuries of Hearing Organ
452	Kinney	Acoustic Trauma
454	Kos	Clinical Orientation in Management of Dizziness
458	Lewin	Facial & Nasal Reconstructive Surgery
465	Moore	Diseases of Maxillary Sinus
469	Pastore	A Lump in the Throat
473	Pressman	Rhinologist & Problem of Rhinoplasty
474	Putney	Surgery of Neck Tumors
478	Roberts, G. J.	Management of Nasal Fractures
482	Seltzer	Plastic Surgery of Nose & Face-Lift Operation
484	Shambaugh	Failures in Fenestration Surgery
494	Van Alyea	Chronic Sinusitis
498	Panel	Head Pain
500	Panel	Tumors & Infections of Neck
502	Panel	Medications

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Previous to OCTOBER 1

Send order to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

After OCTOBER 1, mail to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
c/o Palmer House, Chicago, Illinois

Academy Candidates and Guests May Purchase Tickets at Registration Desk Only

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 10

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

☐ Monday

☐ Tuesday

☐ Wednesday

☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1952 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD	FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
	Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON. 4						
P.M. 5						
6						
TUES. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
WED. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
THURS. 1						
A.M. 2						
3						
P.M. 4						
5						
6						

News - - - Notes

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

There will be a scientific meeting in conjunction with La Ligue Contre le Trachome on Monday, June 23, 1952, at 2:30 p.m. at the Institut des Jeunes Aveugles, 56 Boulevard des Italiens, Paris.

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY

Dr. Luis Sanchez Bulnes, General Secretary for the IV Congress of the Pan-American Association of Ophthalmology, recently held in Mexico City, reports that the proceedings of the Congress will be ready for distribution early in June. There will be three volumes of about 500 pages each, and most of the material will be in English. Ophthalmologists not attending the Congress who are interested in buying these proceedings may purchase them at $20 per copy. All inquiries should be addressed to Dr. Luis Sanchez Bulnes, Gomez Farias, 19, Mexico, D. F., Mexico.

AMERICAN ORTHOPTIC COUNCIL

The American Orthoptic Council announces that its annual intensive course in Orthoptics will be presented in St. Louis, Missouri, from July 1, 1952 through August 30, 1952. The tuition is $150.00, of which $50.00 is payable at the time of application as a matriculation fee. The Council requires a minimum of nine months for certification and those taking the course receive credit for two months. The remainder of the training time is spent in practical work similar to an internship at any one of a number of approved training places. All practical training must be completed under the supervision of an orthoptic technician who has received the Council's Certificate. The Council endeavors to place all students for practical training if they have not already made such arrangements prior to taking the course. Because of administrative reasons, few training places will accept students for training for periods of less than nine months.

Inquiries for further information or for applications may be addressed to Richard G. Scobee., M.D., American Orthoptic Council, 640 S. Kingshighway Blvd., St. Louis 10, Mo.

NATIONAL COUNCIL TO COMBAT BLINDNESS

The National Council to Combat Blindness, in accordance with its program of supporting ophthalmological research, is now welcoming applications for its third annual grant-in-aid awards, to be made in June of 1952. Applications to be considered for this group of awards should be in the office of the Council no later than April 15, 1952. Application forms may be obtained by addressing: Secretary, National Council to Combat Blindness, 1186 Broadway, New York 1, N. Y.

The organization's Committee on Research includes Dr. Phillips Thygeson, University of California, Berkeley, Chairman; Dr. Alson E. Braley, State University of Iowa College of Medicine, Iowa City; Dr. Dan M. Gordon, Cornell University Medical College, New York; Dr. Stuart Mudd, University of Pennsylvania School of Medicine, Philadelphia; Dr. Charles A. Perera, Columbia University College of Physicians and Surgeons, New York; Dr. Samuel L. Saltzman, New York Medical College, Flower and Fifth Avenue Hospitals; and Dr. James H. Allen, Tulane University, New Orleans.

In 1951 the following grants-in-aid were awarded: Retina Foundation, Massachusetts Eye and Ear Infirmary, Boston, $5,000, Dr. Endre A. Balazs, acid mucopolysaccharides in the vitreous body, their role in the composition of the vitreous body and their relationship to the proteins in the vitreous gel; University Hospitals, University of Iowa, Iowa City, $2,700, Dr. Hermann M. Burian, study of the electrical response of retina and brain in patients with amblyopia ex anopsia and suppression; New York University Post Graduate Medical School, New York, $1296, Dr. Helena Fedukowicz, attempts to grow the virus of inclusion conjunctivitis, follicular conjunctivitis and folliculosis; New York Eye and Ear Infirmary, New York, $1600, Dr. Louis J. Girard, focal infection clinic; State University of Iowa Medical School, Iowa City, $1200, Dr. Ralph C. Janes, a study of the incidence and basic cause of cataract, hemorrhages, and degenerative changes in the retina and optic nerve of alloxan diabetic animals; University of California

Medical School, Berkeley, $1200, Dr. Samuel J. Kimura, the relationship of uveitis to bacterial allergy; University of Pennsylvania, Philadelphia, $2500, Dr. H. P. Kirber, virus infections of ocular tissues grafted onto the choricallantoic membrane of the chick embryo; Government Hospital, Haifa, Israel, $2500, Dr. I. C. Michaelson, factors affecting new vessel growth into the cornea; Indiana University Medical School, Indianapolis, $1500, Dr. T. F. Schlaegel, Jr., studies on the mechanism of development of contralateral granulomatous uveitis from the use of horse serum in rabbits; New York Hospital, Cornell Medical Center, New York, $750, Dr. Edward Tolstoi, retinopathy in diabetes; New York Medical College, Flower and Fifth Avenue Hospitals, New York, $466, Dr. Charles Haig, continuance of study on regional light sensitivity of the retina.

INDIANA

The Indiana Academy of Ophthalmology and Otolaryngology will hold its annual meeting in South Bend, Ind., on the campus of the University of Notre Dame, April 30 and May 1, 1952. The guest speakers will include Dr. G. O'Neil Proud, Kansas City, Kan., and Dr. Harold G. Scheie, Philadelphia.

All physicians practicing ophthalmology or otolaryngology are welcome. For further information contact Dr. John R. Swan, Secretary-Treasurer, 23 E. Ohio St., Indianapolis 4, Ind.

IOWA

The Department of Ophthalmology, College of Medicine, of the State University of Iowa, Iowa City, has announced the addition to their staff of associate professor, Dr. Hermann M. Burian of Boston.

MISSOURI

Dr. Harry N. Glick, St. Louis, received the first award for his scientific exhibit, "The Anatomy and Surgery of the Temporal Bone," at the III Pan-American Congress of Otolaryngology and Broncho-Oesophagology, which was held in Havana, Cuba, January 20-24, 1952. Dr. Glick is assistant professor of clinical otolaryngology at Washington University Medical School, St. Louis.

The Department of Ophthalmology of Washington University's School of Medicine is in the process of forming its 1952-1953 class for the Basic Course in Ophthalmology. The next session begins September 15, 1952 and ends May 15, 1953. Tuition is $800 and the course is approved by the Veterans Administration. Instruction includes 393 hours of didactic work and 759 hours of clinical work. With the clinical background gained during the course, the average student can usually take full advantage of a residency and will require close supervision chiefly for surgical work. The course is limited to 12. All inquiries or applications should be addressed to Richard G. Scobee, M.D., Director of Graduate Training in Ophthalmology, 640 S. Kingshighway Blvd., St. Louis 10, Mo.

MONTANA

The summer meeting of the Montana Academy of Oto-ophthalmology will be held in Missoula on Friday, September 19, 1952. Dr. John Chenault Long, Associate Clinical Professor of Ophthalmology at the University of Colorado School of Medicine, will be the visiting speaker.

OREGON

The third annual John E. Weeks Memorial Lecture in Ophthalmology was given on March 27, 1952 during the Thirteenth Annual Spring Convention in Ophthalmology and Otolaryngology of the Oregon Academy of Ophthalmology and Otolaryngology and the University of Oregon Medical School by Dr. Frederick C. Cordes, San Francisco. His subject was "The Visual Prognosis in Diabetes."

PENNSYLVANIA

The Eighth Annual Joint Meeting of the Diplomates Association of Berks County Physicians and the Reading Eye, Ear, Nose and Throat Society was held on February 20, 1952, at the Wyomissing Club, Reading, Pa.

The Speaker of the evening was Dr. Howard T. Karsner, Medical Research Advisor to the Bureau of Medicine and Surgery, Department of the Navy, Washington, D. C. Dr. Karsner spoke on the subject of "Modern Directions of Medical Investigation," with special attention to atomic radiation.

Dr. Irving H. Leopold has been appointed an attending surgeon at the Wills Eye Hospital, Philadelphia, succeeding to the service of the late Dr. Louis Lehrfeld, who died on February 6, 1952. Dr. Lehrfeld had served the Wills Eye Hospital

over a period of thirty-six years until his death.

The Fifteenth Annual deSchweinitz Lecture will be given on Thursday, November 20, 1952, by Dr. Francis Heed Adler of Philadelphia. His subject will be "The Pathologic Physiology of Strabismus."

BRAZIL

The VIII Brazilian Congress of Ophthalmology is scheduled to convene in Sao Paulo in July 1954. The Executive Committee of this Congress is comprised of: Prof. Cyro de Barros Rezende and Prof. Moacyr E. Alvaro, Presidents; Dr. Sylvio de Almeida Toledo, Dr. Rubens Belfort Mattos, Dr. Paulo Braga Magalhaes, General Secretaries; and Dr. Penido Burnier Filho, Treasurer.

FRANCE

The Societe Francaise d'Ophtalmologie will hold its annual Congress June 22-26, 1952. The chief address will be presented by Dubois-Poulsen on "Perimetry." Other papers will discuss neuro-ophthalmology, the uvea, the retina, and glaucoma.

The Societe d'Ophtalmologie de Paris meets on the third Saturday of every month at 9:30 a.m. in the small Amphitheater of the Faculte de Medicine de Paris, 12 rue de l'Ecole de Medicine, excepting in July, August and September. In November there will be an important meeting which will be held on the third Sunday of the month. Dr. Jean Gallois will present his report on "Vascular Medications in Ophthalmology," and there will be a discussion of modern illumination.

A group of seven ophthalmological societies in France is known as "Societes d'Ophtalmologie de France." This group meets once a year in one of the large French cities. A report is presented together with free papers. In 1952 the annual meeting was held in Nantes, April 5 and 6. The report was read by Prof. Amsler (Zurich) on "Chronic Uveitis."

ITALY

A National Congress is being planned by the Societa Oftalmologica Italiana to be held in Turin, October 6-9, 1952. The official program will include a paper on "Ocular Manifestations of the Virus and Rickettsia" by Professors V. Cavara and G. B. Bietti; "Physiopathology and the Clinical Treatment of Oculo-Encephalitis" by Professors B. Alajmo and A. Rubino; and "Etiopathogenesis and Therapy of Hydrophthalmos" by Professors R. Gallenga and Matteucci.

THE NETHERLANDS

A meeting of the Nederlandsch Oogheelkundig Gezel schap te Amsterdam will be held September 20-21, 1952, at Groningen. Dr. Jonas S. Friedenwald, Baltimore, will present the Donders Lecture at this meeting.

POSITIONS AVAILABLE

A veteran, 30 years of age, married with one child, is completing resident training in ophthalmology at the Manhattan Eye, Ear and Throat Hospital. His training includes: (1) Nine months of ophthalmology in U. S. Navy under an ophthalmologist certified by the Board; (2) Nine-month basic science course at New York University; (3) Nine-month preceptorship with Dr. Truman L. Boyes; (4) Twenty-seven months of resident training at Manhattan Eye, Ear & Throat Hospital. He is interested in locating an association or partnership with another ophthalmologist or with an otolaryngologist. For further information please write to Edward Peter Perley, M.D., 524 East 20th Street, New York, N. Y.

Approved residency in Otolaryngology at the Veteran's Hospital, Kingsbridge, Bronx, N. Y., will become available July 1, 1952. It is for a three-year period with a salary of $2400 yearly to start. Teaching staff, endoscopy, rhinoplasty, head and neck surgery, basic sciences. For details write to David H. Jones, M.D., 140 East 54th St., New York 22, N. Y.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse or guarantee any of the published listings.

Residency in Ophthalmology beginning July 1, 1952 at the George F. Geisinger Memorial Hospital and Foss Clinic, Danville, Pa. Applicant must have had previous ophthalmic training and/or postgraduate course in ophthalmology. Statistics for 1951: Out patients, 11,192; refractions, 5,480; consultations, 394; ophthalmic operations, 362; glasses released, 3,551; cataract extractions, 78; ophthalmic admissions, 285. Applications should be addressed to Clyde H. Jacobs, M.D., Director of the Department of Ophthalmology, Foss Clinic, Danville, Pa.

Information regarding Research Fellowships in Ophthalmology at Indiana University Medical Center may be obtained by writing to Dr. T. F. Schlaegel, Jr., 1040 West Michigan Street, Indianapolis 7, Ind. It is not necessary to have had training in ophthalmology to be accepted for these positions. For those who have not had a graduate course in ophthalmology, these research fellowships will provide some training prior to a residency. The fellows assist in the eye clinic and at eye surgery and attend the formal classes for eye residents. The stipend is $150.00 per month plus room and laundry.

Approved three-year residency in Otolaryngology and Broncho-Esophagology at the Geisinger Memorial Hospital. Three year teaching program includes the Basic Science Course in Otolaryngology at the University of Pennsylvania with tuition paid by the Hospital. For details write to F. W. Davison, M.D., Geisinger Hospital, Danville, Pa.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France
Secretary-General: Prof. A. Franceschetti, rue A—Jentzer 22, Geneva, Switzerland

International Council of Ophthalmology

President: Prof. F. W. Nordenson
Secretary: Dr. Holger Ehlers, Rigshospital, Copenhagen, Denmark

International Organization Against Trachoma

President: Dr. A. F. MacCallan
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland

Pan-American Association of Ophthalmology

President: Dr. M. E. Alvaro
Secretary: Dr. Thomas D. Allen, 122 South Michigan Avenue, Chicago 2, Ill.
Time and Place: February 1956, Santiago, Chile

Pan-American Association of Ophthalmology, Puerto Rico Chapter

President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Jose C. Gros
Secretary: Dr. Pedro Hernandez Gonzalo, Calle 8, 358, Havana, Cuba

FOREIGN

The Ophthalmological and Otolaryngological Society of Alberta

President: Dr. W. E. Spankie
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: Spring of 1953 in Edmonton

All-India Ophthalmological Society

President: Dr. G. Zachariah
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, Queen's Road, Bombay 4, India

Associacao Medica do Instituto Penido Burnier

President: Dr. Leoncio de Souza ueiroz
First Secretary: Dr. Milton B. de Toledo
Second Secretary: Dr. Aloisio Af. Ferreira

Austrian Otolaryngological Society, Vienna

President: Prof. Dr. E. Schlander
Secretary: Doz. Dr. O. Novotny, Vienna IX, Alserstrasse 4, Austria
Time and Place: Annually

Belgian Ophthalmologic Society

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, Avenue Ruelens 179, Louvain
Time and Place: January, June and November. Bruxelles

Bombay Ophthalmologists' Association

Chairman: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

British Association of Otolaryngologists

President: Mr. V. E. Negus
Honorary Secretary: Mr. F. C. W. Capps, 45 Lincoln's Inn Fields, London W. C. 2, England

British Medical Association, Section on Ophthalmology

President: Mr. O. G. Morgan
Secretary: Mr. A. G. Cross, 27 Harley Street, London W. 1, England

Centro de Estudos de Oftalmologia de Sao Paulo

President: Dr. Rubens Belfort Mattos
Secretary: Dr. Jose Carlos Gouvea Pacheco, Marconi 34, Sao Paulo

Chengtu Ophthalmological Society

President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

Chinese Ophthalmology Society

President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

Chinese Ophthalmological Society of Peking

President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

Deutsche Ophthalmologische Gesellschaft Heidelberg

President: Prof. Dr. med. K. Wessely
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik

Faculty of Ophthalmologists

President: Mr. O. M. Duthie
Secretary: Mr. J. H. Doggart, F.R.C.S., 45 Lincoln's Inn Fields, London, W.C. 2, England

French Society of Ophthalmology

Secretary: Dr. Edward Hartmann, 2 Ave Ingres, Paris 16, France

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Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. W. Lohlein
Secretary: Prof. E. Engelking, Heidelberg

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY

President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY

President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

ISRAEL OPHTHALMOLOGICAL SOCIETY

President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. F. A. Anderson
Secretaries: Dr. P. Jameson Evans, 51 Calthorpe Road, Edgbaston, Birmingham 15, England
Dr. R. D. Weeden Butler, 18 Highfield Road, Edgbaston, Birmingham 15, England
Place: Birmingham and Midland Eye Hospital, Church Street, Birmingham 3, England

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP

President: Prof. A. W. Mulock Houwer
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, the Hague

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Mr. A. McKie Reid
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle-on-Tyne, Liverpool, Sheffield and Bradford

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. K. Fuller
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. G. A. Brew
Secretary: Dr. Arnold L. Lance, 135 Macquarie Street, Sydney, New South Wales
Time and Place: August 1953, in Melbourne

OPHTHALMOLOGICAL SOCIETY OF COPENHAGEN

President: Prof. Dr. Holger Ehlers
Secretary: Dr. Carl Johan Moellenbach, Rigshospitalets oejenafdeling, Copenhagen O, Denmark
Place: Rigshospitalets oejenafdeling

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Dr. Ibrahim Ahmad Mohammad
Secretary: Dr. Mahmoud Lutfi, Ophthalmic Hospital, Giza
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah, 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ

Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, México, D. F.
Time and Place: Second Friday of each month. Hospital de Neustra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND

President: Dr. L. S. Talbot
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA

President: Dr. L. Staz
Secretary-Treasurer: Dr. J. Guillaume Louw, 901 Dumbarton House, Adderley Street, Cape Town
Time: Annually

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. M. H. Whiting
Honorary Secretaries: Mr. A. J. B. Goldsmith and Mr. Harold Ridley, 45 Lincoln's Inn Fields, London

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN

President: K. Lindner
Secretary: Ch. Eyb, Wien, IX, Alserstrasse 4

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. F. A. Anderson
Hon. Secretary and Treasurer: Ian C. Fraser, F.R.C.S., Red Roofs, Kingsland, Shrewsbury

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Jesus Eusebio
Secretary-Treasurer: Dr. Jesus Tamesis, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY

President: Prof. Dr. W. Kapuscinski
Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Montague Hine, F.R.C.S.
Secretaries: Arthur Lister, F.R.C.S., 56 Wimpole Street, London, W.1., England
H. E. Hobbs, F.R.C.S., 129 Harley Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY
President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB
President: Dr. E. H. Cameron
Secretary: Dr. John Marshall, 11 Clairmont Gardens, Glasgow, C.3, Scotland
Time and Place: Last Saturday of March and October, Edinburgh and Glasgow, in rotation

SOCIEDAD ARGENTINA DE OFTALMOLOGIA
President: Dr. Jorge Malbran
Secretary: Dr. Jose Maria Roveda, Lezica 4472, Buenos Aires
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA
President: Dr. Italo Martini
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORRINOLARINGOLOGIA
President: Dr. Jorge Suarez-Hoyos, Carrera 5a, no. 13-39, Bogota, Colombia
Secretary: Dr. Francisco Arango
Time and Place: Second Tuesday of each month. Club Medico

SOCIEDAD CUBANA DE OFTALMOLOGIA
President: Dr. Oscar F. Horstmann
Secretary: Dr. Heriberto Buch Granados, Calle B No. 668 ent. 27 y 29, Vedado, Habana, Cuba
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA
President: Dr. Teodulo M. Agundis
Secretary: Dr. Jose Luis Arce, Viena 3-5, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD NICARAGUENSE DE OFTALMOLOGIA
President: Dr. Ricardo Lacayo
Secretary: Dr. Gilberto Cuadra, Clinica Especializada, Managua, D. N., Nicaragua, C. A.

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA
Chairman: Dante J. Yadarola
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA
President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jal.
Place: Edificio Lutencia Despacho 101

SOCIEDAD OFTALMOLOGICA HISPANO AMERICANA
President: Dr. Soria
Secretary: Dr. Costi, 3. Calle Montalban, Madrid

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL
President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDAD OFTALMOLOGICA DE VALPARAISO
President: Prof. Dr. Jean H. Thierry
Secretary-Treasurer: Dr. Jorge Oyarzun O, Hospital de Ojos, Calle Blas, Cuevas, Valparaiso

SOCIEDAD PERUANA DE OTO-RINO-LARINGOLOGIA Y OFTALMOLOGIA
President: Dr. Enrique Cipriani V., Pasaje Olaya 156, Lima, Peru
Secretario General: Dr. Enrique Haro
Secretario de Actas: Dr. Alberto Castillo R.

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA
President: Dr. Paulo C. Pimental
First Secretary: Dr. Pedro Moacyr de Aguiar, Ladeira Santa Thereza 136, Apt. 101, Rio de Janeiro
Second Secretary: Dr. Luiz Eurico Ferreira
Time: Third Friday of every month from April to December

SOCIEDADE DE OFTALMOLOGIA DEL NORTE
President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS
President: Dr. Amelio Bonfioli
Secretary: Dr. Mariana Noronha, Pauso Alegre 1150, Belo Horizonte, Est. Minas Geraes

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA
President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIEDADE DE OFTALMOLOGIA E OTO-RINO-LARINGOLOGIA DO RIO GRANDE DO SUL
President: Dr. Mario Araujo Azambuja
First Secretary: Dr. Humberto Lubisco, Rua Donna Laura 45, Porto Alegre, Est., Rio Grande do Sul
Second Secretary: Dr. Ivo Kuhl

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO
President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paulo Aranha de Azevedo, Xavier de Toledo 71,1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO
President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIETA OFTALMOLOGICA ITALIANA
President: Prof. Giuseppe Ovio
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE
President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time: Last Sunday of February, June and November

SOCIETE D'OTO-RHINO-LARYNGOLOGIE
Administrateur: G. Raimond, 17et19, Rue de Buci, Paris (VI.) France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS
President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm, Sweden

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND
President: Campbell Shaw, M. R. C. S.
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, England

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. James Milton Robb
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 12-17, 1952, Chicago

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES
President: Dr. M. E. Alvaro
Secretary-Treasurer: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

AMERICAN LARYNGOLOGICAL ASSOCIATION
President: Dr. Gordon B. New
Secretary: Dr. Louis H. Clerf, 1530 Locust, Philadelphia 2, Pa.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.
President: Dr. C. Stewart Nash
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.

EASTERN SECTION
Vice-President: Dr. Francis W. Davison
Secretary: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.

MIDDLE SECTION
Vice-President: Dr. Ralph J. McQuiston
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill.

SOUTHERN SECTION:
Vice-President: Dr. Lester A. Brown
Secretary: Dr. James W. McLaurin, Raymond Building, Baton Rouge 6, La.

WESTERN SECTION:
Vice-President: Dr. Howard P. House
Secretary: Dr. Harold Boyd, 1136 West Sixth Street, Los Angeles, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY
Chairman: Dr. A. Ray Irvine
Secretary: Dr. Trygve Gundersen, 101 Bay State Road, Boston, Mass.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY
Chairman: Dr. James M. Robb
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis 3, Tenn.

AMERICAN OPHTHALMOLOGICAL SOCIETY
President: Dr. Lawrence T. Post
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th Street, New York 19, N. Y.

AMERICAN OTOLOGICAL SOCIETY
President: Dr. Kenneth Day
Secretary: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY
President: Dr. Norman N. Smith
Secretary: Dr. Joseph G. Gilbert, 1404 President Street, Brooklyn 13, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY
President: Dr. William H. Evans
Secretary-Treasurer: Dr. Joseph W. Hampsey, 806 May Building, Pittsburgh 22, Pa.
Time and Place: Annual meeting, October 17, 1952, Chicago

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.
Chairman: Dr. Walter H. Fink
Secretary-Treasurer: Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Dr. R. G. C. Kelly
Secretary: Dr. J. Clement McCulloch, 380 Medical Arts Building, Toronto, Ontario

CANADIAN OPHTHALMOLOGICAL SOCIETY
President: Dr. R. F. Nicholls
Secretary: Dr. J. F. A. Johnston, 174 St. George Street, Toronto, Ontario

CANADIAN OTOLARYNGOLOGICAL SOCIETY
President: Dr. Jules Brahy
Secretary: Dr. W. Ross Wright, 361 Regent Street, Fredericton, New Brunswick

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.
President: Mr. Mason H. Bigelow
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY
President: Dr. Bryant P. Smith
Secretary: Dr. Frank L. Bryant, 2622 Greenwood, Shreveport, La.
Time and Place: First Monday of month, September through June. Shreveport Club

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY
Chairman: Dr. John P. Frazer
Secretary: Dr. C. W. Trexler, Young Hotel Building, Honolulu, T. H.
Time and Place: Each Thursday (dinner meetings) 6:30 p.m., Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Harry O. Frazier
Secretary: Dr. Robert L. Rees, 115 East South Temple Street, Salt Lake City, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: William B. Clark
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.

NASSAU OPHTHALMOLOGICAL SOCIETY
President: Dr. A. Milton Goldman
Secretary: Dr. Ward L. Mould, 131 Fulton Avenue, Hempstead, N. Y.
Time and Place: Fourth Monday in February, April, September and November at 7:30 p.m. at Meadowbrook Hospital, Hempstead, N. Y.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Benjamin A. Sachs
Secretary: Dr. Garrett L. Sullivan, 101 Bay State Road, Boston 15, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Walter B. Hoover
Secretary-Treasurer: Dr. Burton E. Lovesey, 76 Bay State Road, Boston 15, Mass.
Time and Place: Quarterly. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Lewis F. Morrison
Secretary-Treasurer: Dr. Howard P. House, 1136 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 1952. Salt Lake City, Utah

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William B. Hubbard
Secretary: Dr. Frank A. Ware, 514 Genesee Bank Bldg., Flint, Mich.
Time and Place: Second Tuesday evening of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY EYE AND EAR ACADEMY
President: Dr. James Reeder, Jr.
Secretary-Treasurer: Dr. W. P. Davey, 627-632 Frances Building, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edley H. Jones
Secretary: Dr. F. A. Holden, Medical Arts Building, Baltimore, Maryland
Time and Place: November 10-13, 1952, Miami, Florida

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. C. C. Cooley
Secretary: Dr. J. G. Sellers, 413 Medical Arts Building, Norfolk, Virginia

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. James K. Trumbo
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.

STATE

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Eugene Calloway, Sr.
Secretary-Treasurer: Dr. Karl B. Benkwith, Catoma Street, Montgomery 5, Ala.

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. C. G. Hinkle
Secretary: Dr. K. W. Cosgrove, 113 East Capitol Avenue, Little Rock, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. George F. Keiper, Jr.
Secretary: Dr. Maurice W. Nugent, 2007 Wilshire Blvd., Los Angeles 5, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. J. Leonard Swigert
Secretary: Dr. James C. Strong, 227 Sixteenth Street, Denver, Colo.
Time and Place: Every third Saturday. Colorado Medical Center

COLORADO OTOLARYNGOLOGICAL SOCIETY
President: Dr. Ivan E. Hix
Secretary: Dr. Merrill O. Dart, 3191 S. Broadway, Englewood, Colo.
Time and Place: First Saturday of each month, October through May. Colorado University School of Medicine

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Charles C. Grace
Secretary-Treasurer: Dr. Carl S. McLemore, 1202 South Kuhl Avenue, Orlando, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. F. N. Aldrich
Secretary: Dr. W. Eugene Matthews, Augusta, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. L. Van Buskirk
Secretary-Treasurer: Dr. John R. Swan, 23 East Ohio Street, Indianapolis 4, Ind.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Byron Merkel
Secretary: Dr. Carl A. Noe, 601-611 Highley Building, Cedar Rapids, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. N. L. Francis
Secretary: Dr. W. D. Pitman, First National Bank Building, Pratt, Kan.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Lyman Richards
Secretary: Dr. Lawrence R. Dame, 78 Federal Street, Greenfield, Mass.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. William D. Irwin
Secretary: Dr. V. E. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Henry L. Williams
Secretary-Treasurer: Dr. Francis M. Walsh, 3939 West 50th Street, Minneapolis 10, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. Fritz D. Hurd
Secretary-Treasurer: Dr. H. L. Casebeer, Pennsylvania Building, 44 West Park Street, Butte, Mont.
Time and Place: September 19, 1952, Missoula, Mont.

NEW MEXICO OPHTHALMOLOGIC SOCIETY
President: Dr. A. L. Schonberg
Secretary: Dr. Howard Peck, Medical Arts Square, Albuquerque, New Mexico
Time: Meetings four times a year

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. G. M. Billings
Secretary: Dr. MacLean B. Leath, 529 North Main Street, High Point, N. C.
Time and Place: September 15-17, 1952, Hotel Robert E. Lee, Winston-Salem, N. C.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Richard S. Fixott
Secretary: Dr. George E. Chamberlain, 909 Medical Dental Building, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May. Old Heathman Hotel, Portland

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Matthew S. Ersner
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.
Time and Place: May 8-11, 1952, Golden Hall Hotel, Wernersville, Pa.

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Luis J. Fernandez
Secretaries: Dr. Ricardo F. Fernandez, P.O. Box 2206, San Juan 10, Puerto Rico (Ophthalmology)
Dr. Carlos E. Munoz MacCormick, P.O. Box 604, San Juan, Puerto Rico (Otolaryngology)
Time and Place: Bimonthly. Puerto Rico Medical Association Building

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Thomas R. Gaines
Secretary-Treasurer: Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C.
Time and Place: September 15-17, 1952, Hotel Robert E. Lee, Winston-Salem, N. C.

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Sam H. Sanders
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. Charles Dickson
Secretary: Dr. Lyle Hooker, Esperson Building, Houston, Texas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Robert H. Courtney
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh, 104 East Market St., Charlottesville, Va.
Time: May 1952

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Arthur Chandler
Secretary: Dr. Ben W. Bird, 909 Mercer St., Princeton, W. Va.
Time: Biannual

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. William F. Krone
Secretary: Dr. Francis J. Grant, 1224 Salem Avenue, Hillside, N. J.
Time and Place: 8:45 p.m., second Monday of the month. The Academy of Medicine, 91 Lincoln Park, South, Newark

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. R. Anderson
Secretary-Treasurer: Dr. A. L. Peter, 406 Akron Savings and Loan Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January March, May and November. Akron City Club
Time and Place: First Friday and Saturday of March General Oglethorpe Hotel, Savannah, Ga.

ALUMNI ASSOCIATION NEW YORK EYE AND EAR INFIRMARY
President: Dr. W. T. Boland
Secretary-Treasurer: Dr. Hunter Romaine, 111 East 65th Street, New York, N. Y.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. F. Phinizy Calhoun, Jr.
Secretary-Treasurer: Dr. James T. King, 384 Peachtree Street N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION
Chairman: Dr. M. Elliott Randolph
Secretary-Treasurer: Dr. Abraham Kremen, 2355 Eutaw Place, Baltimore 17, Md.
Time and Place: 8:00 p.m., fourth Thursday of every month October to May. Medical and Chirurgical Faculty, 1211 Cathedral St.

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY
President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through May. Thomas Jefferson Hotel

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ASSOCIATION
President: Dr. Joseph Nerbonne
Secretary: Dr. Benjamin Riseman, 41 Bay State Road, Boston 15, Mass.

BROOKLYN OPHTHALMOLOGICAL SOCIETY
President: Dr. Daniel K. Kravitz
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Avenue, Brooklyn 13, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB
President: Dr. Milton A. Palmer
Secretary: Dr. Elizabeth P. Olmsted, 568 Lafayette Avenue, Buffalo 22, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. G. C. Otrich
Secretary-Treasurer: Dr. Alfred G. Schultz, 237 East State Street, Jacksonville, Ill.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Cecil B. Hert
Secretary-Treasurer: Dr. James L. McGraw, 619 University Building, Syracuse, N. Y.

CHATTANOOGA EYE, EAR, NOSE AND THROAT SOCIETY
Chairman and Secretary: Dr. Willard H. Steele, Jr., 552 McCallie Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., fourth Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY
President: Dr. Arthur J. Coombs
Secretary: Dr. Lawrence J. Lawson, 636 Church Street, Evanston, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY
President: Dr. J. Robert Fitzgerald
Secretary-Treasurer: Dr. Gail R. Soper, 636 Church Street, Evanston, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB
Presidium: Dr. Donald J. Lyle, Dr. Karl W. Ascher, Dr. Josef D. Weintraub
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: Second Wednesday night of month, November through May. University Club, Fourth and Broadway

CINCINNATI OTOLARYNGOLOGIC SOCIETY
President: Dr. Raymond L. Hilsinger
Secretary-Treasurer: Dr. Stephen P. Hogg, 556 Doctors' Building, Cincinnati 2, Ohio
Time and Place: 6:30 on the third Monday of each month except July, August, September and December. University Club.

CLEVELAND OPHTHALMOLOGICAL CLUB
President: Dr. G. Leslie Miller
Secretary-Treasurer: Dr. Webb P. Chamberlain, Jr., 7405 Detroit Avenue, Cleveland, Ohio
Time and Place: Second Tuesday of November, January, February and April. Statler Hotel, Cleveland

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY
Chairman: Dr. George F. J. Kelly
Clerk: Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Rex E. House
Secretary-Treasurer: Dr. L. David Wright, Thomas-Spann Clinic, 1546 South Brownlee, Corpus Christi, Texas
Time and Place: First Wednesday of each month. La Louisiane

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. B. Wilkinson
Secretary: Dr. Claude D. Winborn, Medical Arts Building, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. H. McNamee
Secretary-Treasurer: Dr. H. H. Gurau, 213 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL CLUB
Secretary-Treasurer: Dr. Frederick A. Lauppe, 1249 David Whitney Building, Detroit 26, Mich.

DETROIT OPHTHALMOLOGICAL SOCIETY
President: Dr. Arthur S. Hale
Secretary: Dr. Arthur P. Wilkinson, 974 Fisher Building, Detroit 2, Mich.
Time and Place: Fourth Thursday of each month, November through April, at L'Aiglon, Fisher Building

DETROIT OTO-LARYNGOLOGICAL SOCIETY
President: Dr. B. F. Glowacki
Secretary-Treasurer: Dr. John R. Birch, 1010 Maccabees Building, Detroit 2, Mich.
Time and Place: Third Wednesday of each month, September to May. Wayne County Medical Society Building

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION
President: Dr. Harry Miller
Secretary-Treasurer: Dr. Frank Furlong, 713 Union Street, Schenectady, New York
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Hal Maxwell
Secretary: Dr. William Skokan, 1708 Medical Arts Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. St. Joseph's Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION
President: Dr. Lyle Hooker
Secretary: Dr. Claude C. Cody, III, 1304 Walker Avenue, Houston 2, Texas
Time: Second Thursday of each month, October through June

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Marlow W. Manion
Secretary-Treasurer: Dr. Lewis E. Morrison, 603 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.
President: Dr. A. W. McAlester, III
Secretary: Dr. Barnard C. Trowbridge, 411 Alameda Road, Kansas City 2, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Edmund D. Godwin
Secretary: Dr. James V. Keipp, 110 Pine Avenue, Long Beach 2, Calif.
Time and Place: 6:00 p.m., monthly, September through May. Seaside Memorial Hospital

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Victor Goodhill
Secretary: Dr. Orwyn Ellis, 523 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OPHTHALMOLOGY
President: Dr. Everett S. Caldemeyer
Secretary-Treasurer: Dr. J. Spencer Dryden, 1835 Eye Street Northwest, Washington 6, D. C.

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Chairman: Dr. Irvin Feldman
Secretary: Dr. John Louzan, 1801 Eye Street N. W., Washington 6, D. C.
Time and Place: 7:00 p.m., third Tuesday in October, November, January, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis, Tenn.
Time and Place: Second Tuesday in each month, September through May. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. J. P. Wild
Secretary-Treasurer: Dr. Howard High, 324 East Wisconsin Avenue, Milwaukee 2, Wis.
Time and Place: 6:30 p.m., fourth Tuesday of each month, October to May. Athletic Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. Jules Brahy
Secretary-Treasurer: Dr. Leo S. S. Kirschberg, 1390 Sherbrooke Street West, Montreal, Que., Canada
Time: Second Thursday of October, December, February and April

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. John W. Harned, Jr.
Secretary-Treasurer: Dr. Herbert Duncan, Bennie Dillon Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. James W. Smith
Secretary: Dr. A. Gerard De Voe, 30 West 59th St., New York, N. Y.
Time and Place: Third Monday of the month, October through May. New York Academy of Medicine

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Adolph Posner
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24, N. Y.
Time and Place: First Monday of the month, October to May. New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. N. Meador
Secretary: Dr. Rex C. House, 228 Medical-Dental Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., second Monday of each month, October through May. Dusty's, 1739 South Brownlee Street, Corpus Christi, Texas

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. W. McClure
Secretary: Dr. Richard A. Clay, 416 Northwest 13th Street, Oklahoma City, Okla.
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Thomas T. Smith
Secretary-Treasurer: Dr. G. T. Alliband, 1020 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Alfred Cowan
Secretary: Dr. John W. Deichler, 301 South 21st Street, Philadelphia 3, Pa.
Time: First Thursday of each month, November through April

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President: Dr. Thomas F. Furlong, Jr.
Secretary: Dr. John J. O'Keefe, 255 South 17th Street, Philadelphia 3, Pa.

PITTSBURGH ACADEMY OF MEDICINE
President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OTOLOGICAL SOCIETY
President: Dr. Daniel S. DeStio
Secretary: Dr. George C. Schein, 634 Washington Road, Pittsburgh, Pa.
Time and Place: Bimonthly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. H. Frederick Thorlakson
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. William P. Jennings
Secretary: Dr. James E. Landis, 232 North Sixth Street, Reading, Pa.
Time and Place: Third Wednesday of each month, September to July (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Mason Smith
Secretary-Treasurer: Dr. Charles N. Romaine, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Anderson C. Hilding
Secretary: Dr. James P. Tetlie, 626 Medical Arts Building, Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., preceding the monthly St. Louis County Medical Society meeting. St. Mary's Hospital, Duluth

EAR, NOSE AND THROAT CLUB OF ST. LOUIS
Chairman: Dr. Millard F. Arbuckle
Secretary: Dr. Harry N. Glick, 1504 South Grand Boulevard, St. Louis 4, Mo.
Time and Place: Third Wednesday in November, January, March and May. University Club Building

ST. LOUIS OPHTHALMIC SOCIETY
President: Dr. J. M. Keller
Secretary: Dr. Benjamin Milder, 539 North Grand Avenue, St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. W. E. Borley, 655 Sutter Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, except July, August and December. San Francisco County Medical Society Building

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Phil Greene
Secretary: Dr. Cornelius E. Hagan, Jr., 508 Old National Bank Building, Spokane, Wash.
Time and Place: Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB
President: Dr. Theodore Holstein, 523 Medico-Dental Building, Sacramento 14, Calif.
Secretary: Dr. John Berg
Time: Every third Thursday, eight months of the year

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. N. Gardner Mathieson
Secretary: Dr. Robert D. Kiess, 1838 Parkwood, Toledo, Ohio
Time and Place: Third Tuesday of each month, November through May, Toledo Club

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY
Chairman: Dr. Joseph C. Hill
Secretary: Dr. Harold R. Sniderman, 220 Bloor Street West, Toronto 5, Ontario, Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Ralph T. Himes
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of May and October

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY
President: Dr. Vincent Gallizzi
Secretary: Dr. Samuel T. Buckman, 70 South Franklin Street, Wilkes-Barre, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street

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E., E., N., & T. Mo., 26:27, 1947.
Laryngoscope, 56:556, 1946.
New Eng. J. Med., 234:468, 1946.
Annals of Allergy, 4:33, 1946.
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